A STUDY ON FACTORS PREDICTING THE OUTCOME OF PERFORATED PEPTIC ULCER

A Prospective study

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CERTIFICATE

This is to certify that the dissertation titled **"A STUDY ON FACTORS PREDICTING THE OUTCOME OF PERFORATED PEPTIC ULCER"** is the original work done by **DR.T.NANDHAGOPAL**, post graduate in M.S General surgery at the Department of General surgery and Rajivgandhi Government General Hospital, Chennai to be submitted to The Dr.MGR medical university, Chennai towards the partial fulfillment of the requirement for the award of M.S., degree in General surgery, April 2014.

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DECLARATION

I **Dr.T.Nandhagopal** declare that this dissertation titled **"A STUDY ON FACTORS PREDICTING THE OUTCOME OF PERFORATED PEPTIC ULCER"** is a record of the original work done by me in the Department of General surgery and Rajivgandhi Government General Hospital, Chennai during my postgraduate course of M.S General surgery from 2011 to 2013 under the guidance of my unit chief Prof.G.Muralidharan M.S., FICS., FACS., PGDHS and head of the department Prof.S.Deivanayagam M.S., It is submitted to The Dr.MGR medical university, Chennai towards the partial fulfillment of the requirement for the award of M.S., degree in General surgery, April 2014. This record of work has not been submitted previously by me for the award of any degree from any other university.

DR.T.NANDHAGOPAL

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T.Nandhagopal

ABBREVIATIONS

AKI -Acute Kidney Injury

ARDS -Adult Respiratory Distress Syndrome

CT -Computerized Tomography

DU -Duodenal ulcer

GU -Gastric ulcer

H.Pylori -Helicobacter pylori

MODS -Multiple Organ Dysfunction Syndrome

NSAID -Non steroidal Anti Inflammatory Drugs

PPI -Proton pump inhibitor

PPU -Perforated peptic ulcer

PUD -Peptic ulcer disease

USG -Ultrasonagram

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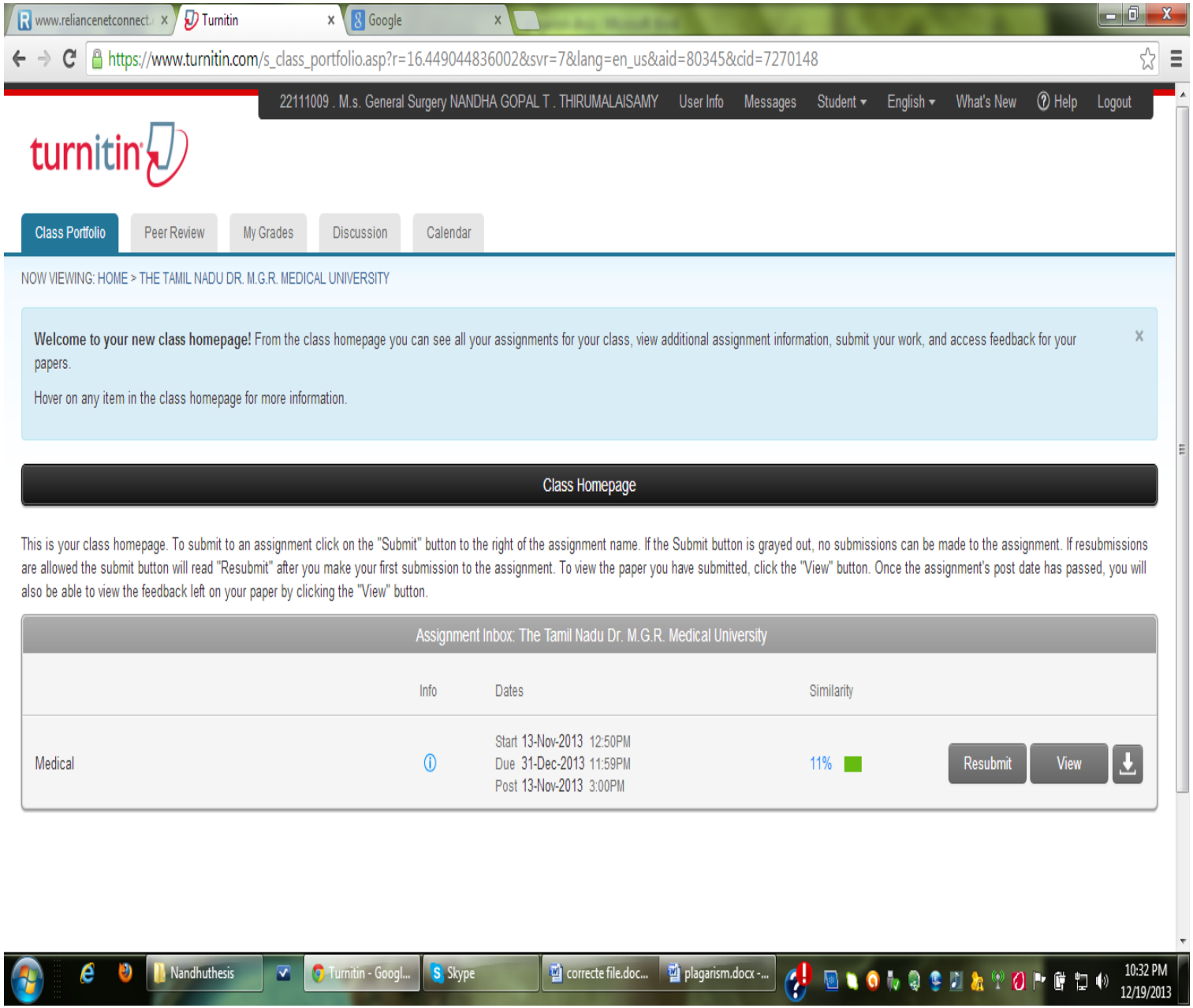
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ABSTRACT

A STUDY ON FACTORS PREDICTING THE OUTCOME OF PERFORATED PEPTIC ULCER

Place: Department of General Surgery, Madras Medical College and Rajivgandhi Government General Hospital, Chennai.

BACKGROUND AND AIM:

With the introduction of H2 receptor antagonists and proton pump inhibitors, the incidence of elective surgery for peptic ulcer (PU) diseases has decreased, although complications of PU such as perforation and bleeding have remained fairly constant. The purpose of this study was to identify the risk factors that predict morbidity and mortality in patients with perforated Peptic Ulcer

METHODS

The following factors were recorded for 100 patients diagnosed and operated for perforated peptic ulcer and were analyzed in terms of morbidity and mortality: age, sex, personal habits of smoking and consuming alcohol, NSAID usage, past peptic ulcer history, co morbid illnesses, the duration of pain, duration between pain and surgery, duration between admission and surgery, surgery duration, shock and American Society of Anesthesiologist score, size, site of perforation, contamination, procedure and complications following the surgery.

RESULTS

Age of 60 years and above had mortality of 46% and also this mortality is high within the first 48 hrs and up to 7 days. Average duration of hospital stay was 7 to 14 days. Female sex was more related to severe disease and mortality (100%). Duration of pain of 3 days had 35.7% and 4 or more 50% mortality. 82% of the patients had past history of peptic ulcer and on and off treatment. 16% of patients had history of NSAID usage before perforation. 48% of patients

had the habit of both smoking and alcohol and 40% are neither smoking nor alcoholic. On physical examination 96% had respiratory distress, 80% had anemia, 38% had tachycardia, 26% both hypotension and tachycardia, and 36% had normal pulse and blood pressure. 94% had signs of peritonitis. 80% showed air under diaphragm in chest X ray. Preoperative lower respiratory infection had 40% mortality. Duration between pain and surgery that is preoperative delay of 48 to 72 hours had 31% and more than 72 hrs had 32% mortality. ASA score of 4 and 5 had 100% mortality and score of 3 had 64%. Gastric perforation (30%) has more mortality than duodenal (20%) perforation. Size of perforation more than 1 cm has 42% mortality. Contamination of more than 1 litre had related to 44% of death. Duration of the surgery more than 2 hours had significant effect on the mortality. 32% were in need of ventilator support and 24% were in need of circulatory support. 76% of patients had adequate renal function. 46% of the patients developed wound infection and 26% had wound dehiscence and underwent secondary suturing. Post operatively 20% patients had lower respiratory infection and 10% had ARDS leading to death. 24% of the patient had acute kidney injury and treated with supportive treatment. 14% of patients had developed multiple organ dysfunctions and died. Sepsis leading to shock and multiple organ dysfunctions is a cause of death.

CONCLUSION

The following factors were associated with morbidity and mortality; Age more than 60 years, duration between initial pain and surgery of more than 48 hours, class III or more shock, ASA score of 3and more, size of more than 1 cm and contamination of more than 1 litre and the following factors were associated with mortality; Preoperative lower respiratory infection, duration of the surgery, Post operative lower respiratory infection, and Acute respiratory distress syndrome.

Key words: perforated peptic ulcer, peptic ulcer perforation.

INTRODUCTION

Perforation is one of the complications of peptic ulcer disease. The development and use of gastric anti secretory agents like H2 receptor blockers, proton pump inhibitors together with the understanding of Helicobacter pylori infection as a cause of disease and its eradication with the drug therapy has resulted in high chance of curing peptic ulcer disease and also preventing recurrence of peptic ulcer.

As a result, there has been a marked decrease in the number of elective surgery for acid reduction in patients with uncomplicated peptic ulcer and there is fall in the incidence of peptic ulcer disease in recent years, globally[1].

Though the number of patients admitted in emergency department for perforated peptic ulcer and emergency surgery for the same has not undergone similar decline, and it remain a significant health problem[1-4].

There is high risk of emergency for perforated peptic ulcer and high rate of postoperative complication which is between 21% to 43%[5, 6]. The morbidity and mortality following the emergency surgery for peptic ulcer perforation and risk factors associated with peptic ulcer perforation were studied by several studies. There are multiple factors which are influencing the outcome of surgery for perforated peptic ulcer. Inspite of plenty of evidence in the literature, the

knowledge about factors which affect the morbidity and mortality that occurs after perforated peptic ulcer is limited.

We have conducted a prospective study in 100 patients admitted for perforated peptic ulcer from January 2013 to October 2013. The rationale of this study is to evaluate the factors that influence the mortality and morbidity in patients operated for peptic ulcer perforation.

AIM OF THE STUDY

The aims of the study are

1. To study the age group, sex, personal habits of smoking and consuming alcohol, NSAID usage, past peptic ulcer history and co morbid illnesses and its relationship with outcome of peptic ulcer perforation.
2. To study the duration of pain, duration between pain and surgery, duration between admission and surgery, surgery duration and its relation to morbidity and mortality.
3. To study the shock and ASA score and effect on the mortality and morbidity in peptic ulcer perforation
4. To study biochemical and hematological factors predicting the final outcome of perforated peptic ulcer
5. To study size, site of perforation, contamination and procedure and mortality and morbidity in peptic ulcer perforation
6. To study the complications following the surgery for peptic ulcer perforation.

REVIEW OF LITERATURE

For thousands of years healthy people have had acute abdominal pain, nausea, vomiting and diarrhoea followed by death in a few hours or days. Often these symptoms were contributed to poisoning and people have been sent to prison for this[7].Henriette-Anne daughter of King Charles I, died suddenly in 1670 at age of 26 after a day of abdominal pain and tenderness. Since poisoning was suspected autopsy was performed which revealed peritonitis and a small opening in the anterior wall of the stomach. However, the doctors had never heard of a perforated peptic ulcer and attributed the opening in the stomach to the knife of the dissector [7, 8]. Necropsies were allowed since 1500 and became more routine between 1600 and 1800 [8, 9]. As a consequence more often perforation of the stomach was observed.

The surgeon Johan Mikulicz-Radecki (1850-1905), often referred to as the first surgeon who closed a perforated peptic ulcer by simple closure said: “Every doctor, faced with a perforated of the stomach or intestine, must consider opening the abdomen, sewing up the hole, and averting a possible inflammation by careful cleansing of the abdominal cavity” [10].

The treatment since has not changed much, still consisting of closure of the perforation primarily by single stitch suture and a convenient tag of adjacent omentum on top of this [11 - 14]. Although this therapy sounds very simple still

PPU remains a dangerous surgical condition, associated with high morbidity and mortality, not to be underestimated [15].

CLINICAL PRESENTATION AND INVESTIGATION

In 1843 Edward Crisp was the first to report 50 cases of PPU and accurately summarized the clinical aspects of perforation; concluding: "The symptoms are so typical, I hardly believe it possible that anyone can fail to make the correct diagnosis." [16].

Patients with PPU have a typical history of sudden onset of acute, sharp pain usually located in the epigastric area and sometimes with referred shoulder pain, indicating free air under the diaphragm [17]. Bases on collected data from 52 papers on PPU clinical characteristics have been summarized in Table 1.

Table 1. *Clinical characteristics of patients with perforated peptic ulcer disease [18, 19, 22, 37, 47-49, 51, 55, 57, 59, 64]*

		Total n=2784
Age (years)	48	n=2328
Male (%)	79	n=2678
History of ulcer (%)	29	n=1140
History of NSAID use (%)	20	n=1109
Smokers (%)	62	n=472
Alcohol use (%)	29	n=198
ASA I (%)	35	n=1120
ASA II (%)	37	n=1060
ASA III (%)	20	n=1060
ASA IV (%)	9	n=1030
Boey 0 (%)	59	n=513
Boey 1 (%)	23	n=513
Boey 2 (%)	16	n=513
Boey 3 (%)	2	n=513
Shock at admission (%)	7	n=1107
Symptoms > 24 hrs (%)	11	n=723
Duration of symptoms (hrs)	13.6	n=837
Free air on x-ray (%)	85	n=510
WBC	12.3	n=147

The typical patient with PPU is male with an average age of 45 years. He may have peptic ulcer disease history (29%), or usage of nonsteroidal anti-inflammatory drugs (NSAIDs) (20%). Nausea and vomiting are present in 50% of cases.

At physical examination pulse rate increased, but seldom goes beyond 90 beats per minute. About 5-10% of patients experience shock with a mean arterial pressure of less than 80 mmHg [18]. Hypotension and high fever are late findings. Obliteration or complete absence of liver dullness was only noted in 37%, so as a diagnostic tool, this has its limitations [13].

In blood analysis a moderate leucocytoses will be found. Main reason for taking a blood sample is excluding other diagnosis like for instance pancreatitis [10]. An X-ray of the abdomen/thorax in standing position will reveal free air under diaphragm in about 80-85 % [13, 19]. Some centres perform abdominal ultrasonography, or abdomen contrast enhanced computerized tomography scans with oral contrast [20]. 80-90% of cases are correctly diagnosed with current radiological techniques [18].

As soon as diagnosis is made resuscitation is started with large volume crystalloids, nasogastric suction to empty the stomach; and administration of broadspectrum antibiotics [19, 21]. When PPU has been diagnosed, there are a few different therapeutic options to be taken into consideration [18]. First of all it must be evaluated if the patients are suitable for surgery or should conservative

treatment be considered instead. If surgery is indicated, is simple closure with or without omentoplasty sufficient or is there a need for definitive ulcer surgery and if there is a need for definitive surgery, which specific operation is indicated? Finally, can the operation be performed laparoscopically or are there risk factors that would made laparotomy a safer option? [18, 22].

PATHOGENESIS

A peptic ulcer is defined as disruption in the integrity and discontinuity of mucosa of duodenum and/or stomach which lead to a local defect with active inflammation. These ulcers are often chronic in nature. Peptic Ulcer Disease includes both gastric and duodenal ulcers.

Ulcers, breaks in the mucosal surface and size >5 mm, with depth extending up to the submucosa. The pathogenesis, diagnosis and treatment are common in both duodenal ulcers (DUs) and gastric ulcers (GUs); but several factors distinguish them from one another.

The pathogenesis of PUD is a complex scenario involving an imbalance between defensive and aggressive factors. The Defensive factors include Mucus-bicarbonate layer, Prostaglandins, Cellular renovation, and Blood flow
The aggressive factors include Hydrochloric acid, Pepsin, Ethanol, Bile salts, Some medications including NSAIDs, etc. [21,] Cocaine[18].

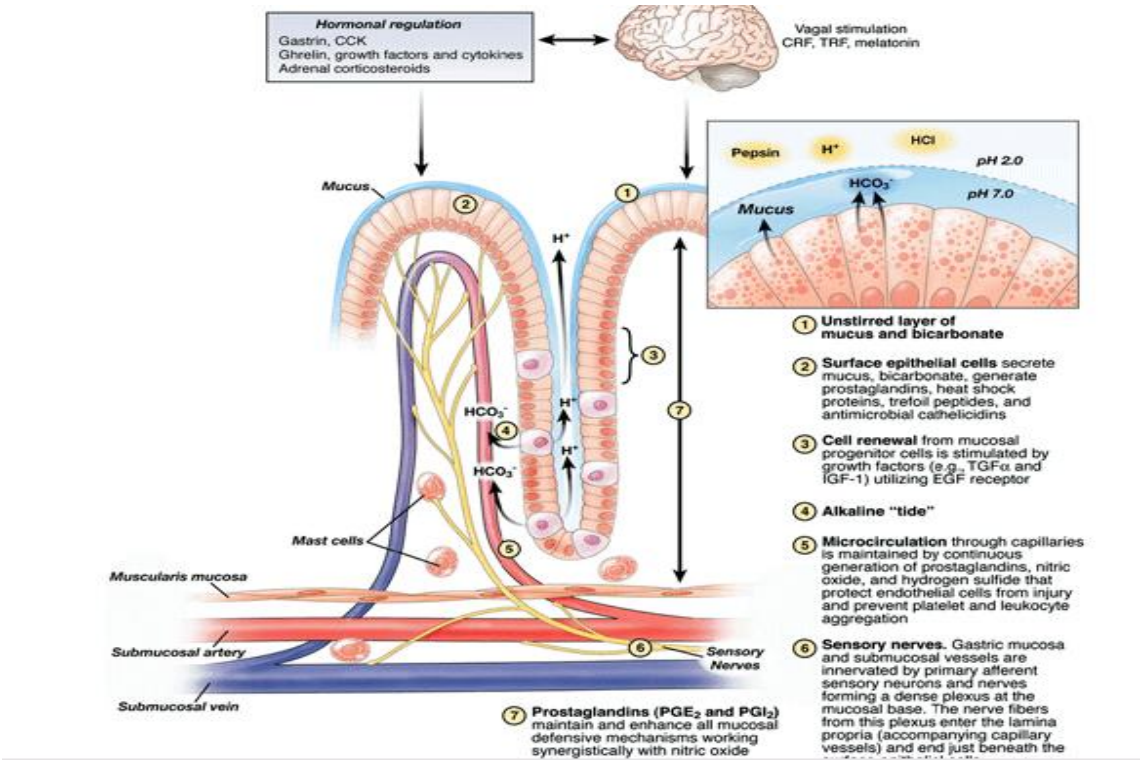
Helicobacter pylori infection and NSAIDs have been identified as the two main causes of peptic ulcer [23] in recent years.

Mucosal defense system

It is a three-level barrier system (Figure 3), composed of

- Pre epithelial,
- Epithelial and
- Sub epithelial elements.

Figure 1



Pre epithelial system

It is a mucus-bicarbonate-phospholipid layer. It is the first line of defense in preventing ulcer formation. This is a physicochemical barrier to multiple molecules, including hydrogen ions protecting the mucosa.

- Mucus is secreted by gastroduodenal surface epithelial cells in a regulated fashion. Contents of mucus are of 95%water and a mixture of mucin a glycoprotein and phospholipids. This mucous gel acts as a nonstirred water layer which impedes diffusion of ions and molecules including pepsin.
- Bicarbonate, secreted by surface epithelial cells of the gastroduodenal mucosa in a regulated manner. Bicarbonate is secreted into the mucous gel. Bicarbonate forms a pH from 1 to 2 at the gastric luminal surface and reaching 6 to 7 along the epithelial cell surface.

Epithelial barrier

Surface epithelial cells provide the next line of defense in protecting the mucosa. They act by producing mucus, bicarbonate and epithelial cell ionic transporters and intracellular tight junctions. These ionic transporters maintain intracellular pH.

Surface epithelial cells generate heat shock proteins that prevent protein denaturation and protect cells from increased temperature, cytotoxic agents, or oxidative stress. Epithelial cells also generate trefoil factor family peptides and cathecidins, which also play a role in surface cell protection and regeneration.

Restitution: when the preepithelial barrier is breached, gastric epithelial cells along the site of mucosal injury can migrate and restore a damaged region. This restitution process occurs independent of cell division. It requires

- Uninterrupted blood flow
- An alkaline pH in the surrounding environment.
- Several growth factors modulate restitution process which include epidermal growth factor (EGF), transforming growth factor (TGF), and basic fibroblast growth factor (FGF),
- Larger defects that are not effectively repaired by restitution require cell proliferation.

Epithelial cell regeneration

This is regulated by prostaglandins and growth factors. Growth factors are EGF and TGF-. During regeneration angiogenesis occurs within the injured micro vascular bed. Both FGF and vascular endothelial growth factor (VEGF) are important regulators of angiogenesis in the gastric mucosa.

Sub epithelial system

It is defense and repair system.

Key component - An elaborate microvascular system within the gastric submucosa

Functions

- Provides bicarbonate to neutralize the acid generated by the parietal cell.
- Provides adequate supply of micronutrients and oxygen
- Removes toxic metabolic by products.

Prostaglandins

Prostaglandins play a central role in defense and repair. The gastric mucosa contains abundant levels of prostaglandins.

Functions

- Regulate the release of mucosal bicarbonate and mucus,
- Inhibit parietal cell secretion
- Maintains mucosal blood flow
- Epithelial cell restitution.

Nitric oxide (NO)

It maintains the gastric mucosal integrity. The key enzyme NO synthase is constitutively expressed in the mucosa which contributes to cytoprotection by

- Stimulating gastric mucus secretion,
- Increases mucosal blood flow and
- Maintains epithelial cell barrier function.

The central nervous system (CNS) and hormonal factors also play a role in regulating mucosal defense through multiple pathways.

There are three clinical phases in the process of PPU[4].

- ***Phase 1***: Chemical peritonitis/ contamination

  The perforation results in chemical peritonitis.

  Gastroduodenal contents are sterilized by acid. In conditions like gastric cancer or when gastric acid is reduced by treatment, bacteria and fungi grow in the stomach and duodenum is present in the peritoneum resulting in contamination.

- ***Phase 2***: Intermediate stage

  There is some spontaneous relief of pain after 6 to 12 hours. This is probably due to the dilution of the gastroduodenal contents leaked in to the peritoneum, irritating in character by ensuing peritoneal exudates.

- ***Phase 3***: Intra-abdominal infection:

  Intra-abdominal infection occurs after 12-24 hrs.

EPIDEMIOLOGY

Perforation occurs in 2-10% of patients with PUD and accounts for more than 70% of deaths associated with PUD. Often perforation is the first clinical presentation of PUD [24]. The incidence of duodenal perforation is 7-10 cases/ 100.000 adults per year. [15, 21, 22, 25-28].

The perforation site usually involves anterior wall of the duodenum (60%), Antrum (20%) and Lesser-curvature gastric ulcers (20%) [25].

Gastric ulcers are associated with higher mortality and a greater morbidity than duodenal ulcers due to haemorrhage, perforation and obstruction [23].

PPU used to be a disorder mainly of younger patients (predominantly males), but recently the age of PPU patients is increasing (predominantly females) [22, 26]. Current peak age is 40-60 years [22].

The need for surgery for PPU has remained stable or even increased and the mortality following peptic ulcer perforation surgery have not decreased since the introduction of H2 receptor antagonists. The peptic ulcers are still responsible for about 20.000-30.000 deaths per year in Europe [25, 29]. This may be due to an increase in use of aspirin and/ or NSAID's [18].

THE ROLE OF HELICOBACTER PYLORI

In 1982 Barry J Marshall and Robin Warren discovered the role of H.pylori in gastric and duodenal ulcers, until this, stress and life style factors were believed to be the most important factor contributing to PUD and PPU [30]. H.pylori infection can be hold responsible [23, 30] for more than 90% of duodenal ulcers and up to 80% of gastric ulcers.

H.pylori infection and the accompanying inflammation decreases antral somatostatin and disrupt the inhibitory control of gastrin release. This is more marked in a cagA-positive strain [25] organism infection. This results in increase

in gastrin release and gastric acid secretion which induces PUD [25]. The infection with H.pylori seems to be acquired in early childhood. The immune system does not contribute to the healing.[9, 23]. H.pylori is not only located on the surface of the gastric mucosa but also in the layer of mucus protecting it causing difficulty in its eradication. The national institutes of Health Consensus Development Panel on Helicobacter pylori in PUD recommended that ulcer patients positive for H.pylori should be treated with antimicrobial agents [31]. The type, number of drugs given and treatment duration differ enormously [31]. Although the problem of antibiotic resistance of H.pylori is increasing, combination therapies can achieve eradication rates of 80% or more [25, 32]. According to the Maastricht III consensus report first line treatment for H.pylori infection should be triple therapy which should compromise a proton pump inhibitor (PPI) plus clarithromycin plus amoxicillin or metronidazole [23, 33.] Monotherapy by just giving antibiotics has proven not to be successful (<30% eradication rate) [23].

Peptic ulcer is diagnosed by upper gastro intestinal endoscopy, but patients do not tolerate well [28]. Carbon 13-urea breath test is expensive, but represents a reliable indicator of H.pylori infection. The preferred method to diagnose H.pylori is by taking peroperative biopsies [28]. Even in patients with PPU and NSAID usage, looking for the presence of H.pylori is advisable, since it can be eradicated easily.

To avoid missing gastric cancer, gastroendoscopy should be performed in patients > 45 yrs with alarming features like weight loss, anaemia, or dysphagia [23].

NSAID

The use of nonsteroidal antiinflammatory drugs attributed to about one of four peptic ulcer perforations and in the elderly it is an important risk factor [62]. Patients concurrently ingesting corticosteroids and NSAIDs had a risk for peptic ulcer disease that was 15 times greater than that of nonusers of either drug [63].

CURRENT MANAGEMENT PPU

Non operative management - conservative treatment

Simple suture open repair technique

Definitive surgery or

Laparascopic surgery

NON OPERATIVE CONSERVATIVE MANAGEMENT

This is known as the Taylor method.

Patients are treated with

- Nasogastric aspiration,
- Antibiotics,
- Intravenous fluids and
- H.pylori triple therapy [29, 32].

Effective gastric decompression and continuous drainage will enhance self healing described by Taylor [15, 31]. Perforations of the stomach were filled up by adhesions to the surrounding viscera which prevented leakage from the stomach into the peritoneum described by Crisp[31]. Since, many reports have been published on this topic, with different success rates [15]. It has been estimated that about 40-80% of the perforations will seal spontaneously and overall morbidity and mortality are comparable [25, 29, 31, 34].

Pre operative delay beyond 12hours after the onset of clinical symptoms will worsen the outcome in PPU [15, 25]. Also in patients > 70 years conservative treatment is unsuccessful with a failure rate as high as 67% [15, 34]. Shock at admission and conservative treatment were associated with a high mortality rate (64%) [15, 29].

Patients can be selected for conservative treatment by performing a gastroduodenogram by Donovan [29]. Non surgical treatment in these patients, who had proven sealing of their perforation site was safe, only resulted in 3% intraabdominal abscess formation and < 2% repeat leak [29].

The advantages of conservative treatment are

- Avoidance of operation with associated morbidity caused by surgery and anesthesia,

- Reduction in formation of intraabdominal adhesion induced by surgery which makes elective surgery for PUD or for other indications in a later phase less complicated and
- Hospital stay might be shorter [35]. However, there are also studies that showed a prolonged hospital stay after conservative treatment [19, 25].

Disadvantages are

- A higher mortality rate in case conservative treatment fails.
- Lack of the benefit of laparoscopy or laparotomy as a diagnostic tool in case the patient was misdiagnosed [34, 35].

Figure 2 : Gastric Antral perforation

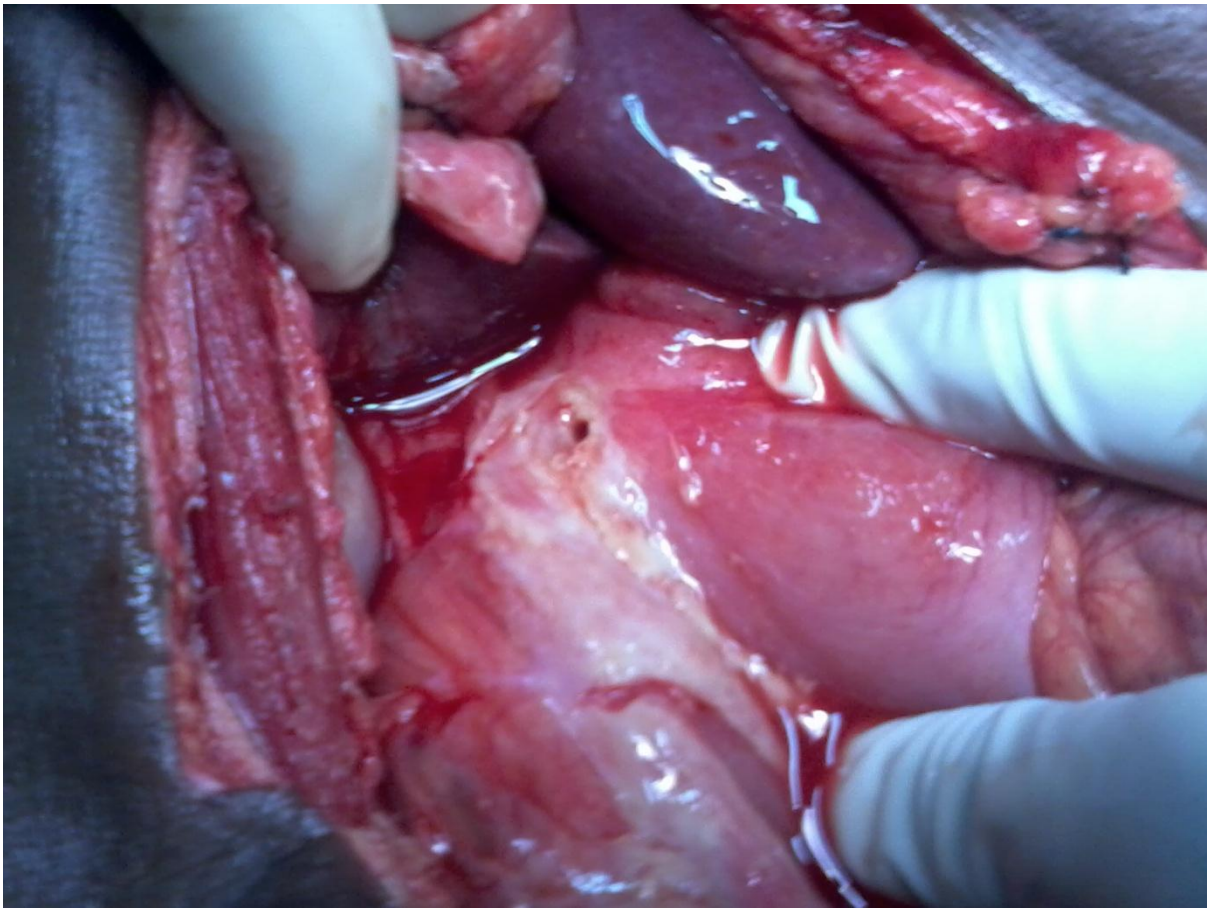
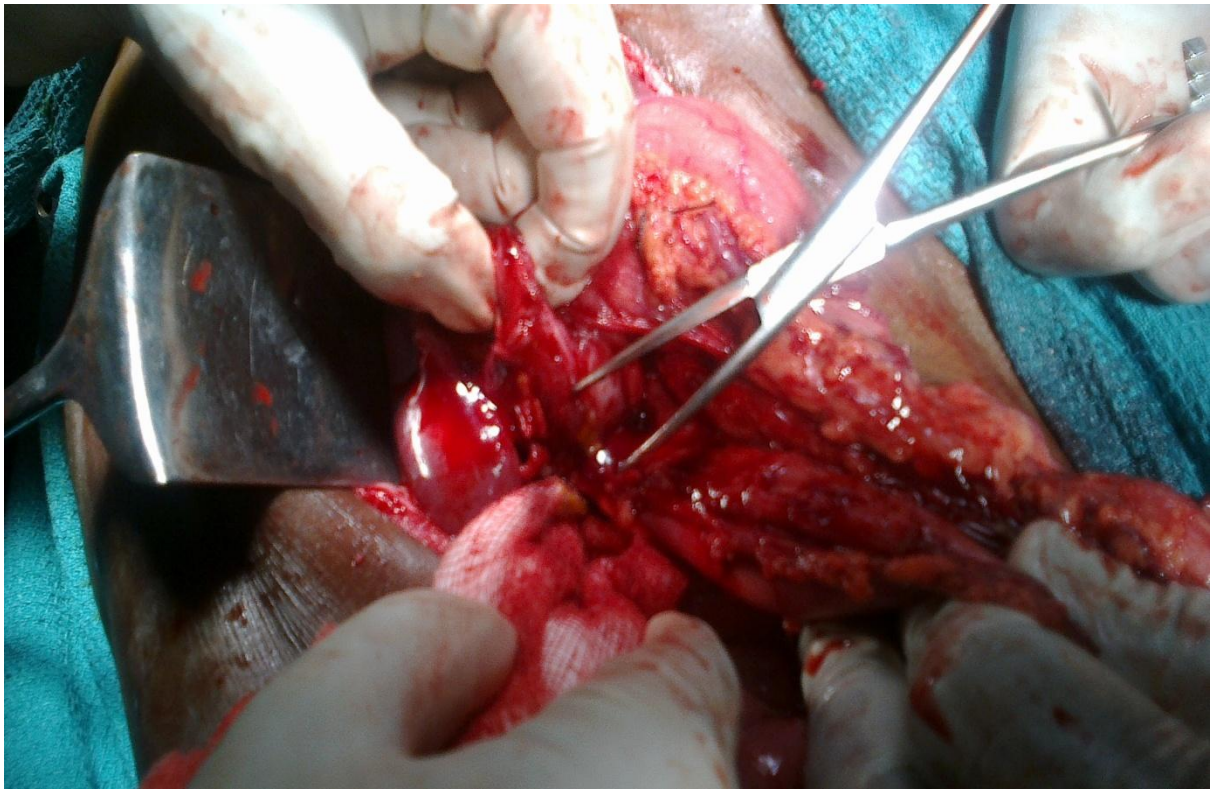


Figure 3 : Duodenum-Part 1 Perforation



Finally one always has to bear in mind that PPU can be a symptom of gastric cancer, so if conservative treatment has been chosen after a few weeks endoscopy should be performed [15, 34].

For conclusion one can say that non operative treatment is limited to patients < 70 years, not eligible for surgical repair due to associated morbidity, with documented contrast studies showing that the perforation has completely sealed. When the patients is in shock or is the time point between perforation and "start treatment" > 12 hours simple closure should be first choice of treatment.

SIMPLE SUTURE OPEN REPAIR TECHNIQUE

All surgical procedures start by giving prophylactic antibiotics at induction of anesthesia. In conventional surgery an upper midline incision is performed. Identification of the site of perforation is not always easy: sometimes a perforation has occurred at the dorsal site of the stomach, only to be detected after opening of the lesser sac through the gastrocolic ligament. Also double perforations can occur. In case of a gastric ulcer a biopsy is taken to exclude gastric cancer.

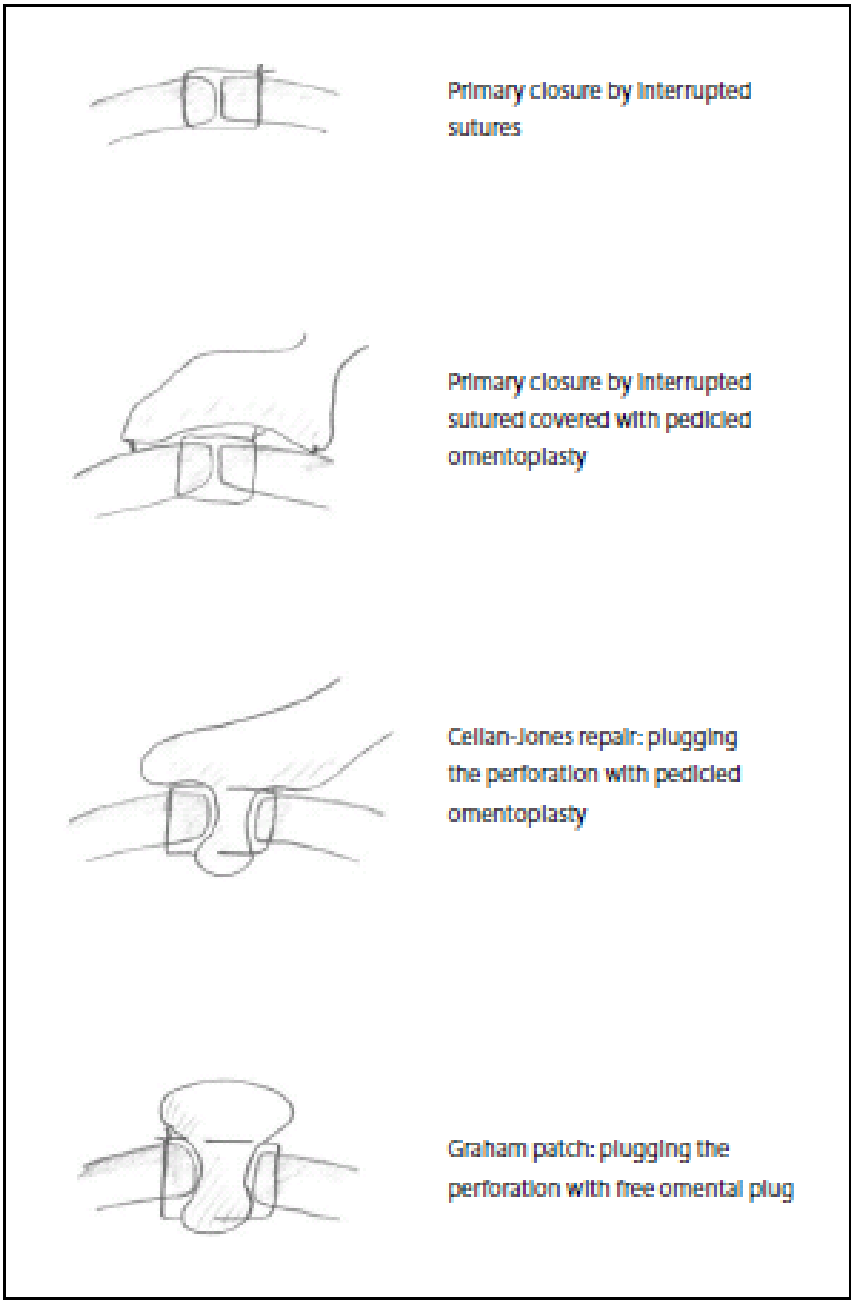
Simple closure of the perforation can be done in different ways (f3igure 4):

1. Simple closure of the perforation by interrupted sutures without omentoplasty or free omental patch,
2. Omentoplasty - Simple closure of the perforation with a pedicled omentum sutured on top of the repair,
3. A pedicled omental plug drawn into the perforation after which the sutures are tied over it and
4. The free omental patch after Graham.

The repair can be tested by either filling the abdomen with warm saline and inflating some air into the nasogastric tube. If no bubbles appear, the perforation has been sealed appropriate. Also dye can be injected through the nasogastric tube[36].

Thorough peritoneal toilet is then performed. A drain is not routinely left [37]. The abdominal wound can be infiltrated with bupivacaine 0.25% at the end of the procedure.

Figure 4: Different suture techniques for closing perforation



Omentoplasty or omental patch: necessary or not? Cellan-Jones published an article in 1929 entitled “a rapid method of treatment in perforated duodenal ulcers”. Treatment of choice at that time was, after excision of friable edges if indicated, the application of purse string sutures and on top an omental graft [38]. An encountered problem was narrowing of the duodenum. To avoid this, he suggested omentoplasty without primary closing of the defect. His technique consisted of placing 4-6 sutures, selecting a long omental strand passing a fine suture through it, the tip of the strand is then anchored in the region of the perforation and finally the sutures are tied off [38]. It was not until 1937 that Graham published his results with a free omental graft [39]. He placed three sutures with a piece of free omentum laid over these sutures, which are then tied. No attempt is made to actually close the perforation [39].

The omental graft provides the stimulus for fibrin formation. His approach has been the golden standard since [40]. Very often surgeons mention they used a Graham patch, but they actually mean they used the pedicled omental patch described by Cellan-Jones [39]. Schein could not have outlined it any clearer: “Do not stitch the perforation but plug it with viable omentum and patch a perforated ulcer if you can, if you cannot, then you must resect” [10].

Irrigation of the peritoneal cavity Although some surgeons doubt the usefulness of irrigation, nothing has been found in literature supporting this theory. General it is one of the most important parts during the surgery

procedure and irrigation with 6-10 litres and even up to 30 litres of warm saline are recommended [22]. However the rational for routinely use of intra-operative peritoneal lavage seems to be more a historical based custom lacking any evidence based support [41].

Drainage or not There seems to be no unanimity of opinion on this topic [22, 36]. In a questionnaire 80% of the surgeons responded that they would not leave a drain [36]. A drain will not reduce the incidence of intraabdominal fluid collections or abscesses [36]. On the other hand the drain site can become infected (10%) and can cause intestinal obstruction [36, 42]. Often a drain is left as a sentinel. However, in case of suspected leakage a CT- scan will provide all the information needed, probably better than a non-productive drain.

DEFINITIVE SURGERY

Indications for elective surgery are still not defined [25]. The number of elective procedures performed for PUD have declined with more than 70% since the 80's [25, 28]. The results of a questionnaire with 607 responders showed that only 0.3% of the surgeons routinely perform a vagotomy for duodenal ulcer complications and 54.5% mentioned they never include it [43].

Reasons for decline in definitive ulcer surgery are:

- Lower recurrence rate of PUD and PPU because of good results of H.pylori eradication and elimination of NSAID use.

- Patients nowadays operated for PPU are older with higher surgical risk which makes them less suitable candidates for definitive ulcer surgery.
- Finally many surgeons practicing today have limited experience with definitive ulcer operations [28].

Patients in whom definitive ulcer surgery should be considered are

- Those with PPU who are found to be H.pylori negative, or
- Those with recurrent ulcers despite triple therapy [18, 25, 31, 44, 45].

In these patients a parietal cell vagotomy is recommended if necessary combined with gastrojejunostomy [46].

LAPAROSCOPY

Since the 90's laparoscopic closure of a perforated peptic ulcer has been described. Laparoscopic surgery offers several advantages.

Advantages

- Minimal invasive diagnostic tool [47].
- Postoperative pain reduction and less consumption of analgesics and
- A reduction in hospital stay [48].
- A reduction in wound infections, burst abdomen and incisional hernia due to shorter scars has been noted [32, 48].
- Avoiding upper laparotomy might lower the incidence of postoperative ileus and chest infections [32, 48].

Drawbacks are

- A prolonged operating time,
- Higher incidence of re-operations due to leakage at the repair site and
- A higher incidence of intra-abdominal collection secondary to inadequate lavage [32, 48, 49].

If the presence of these fluid collections has any clinical relevance is unclear. The higher incidence of leakage might be caused by the difficulty of the laparoscopic suturing procedure. First of all this emphasizes the need for a dedicated laparoscopically trained surgeon to perform this procedure [19]. Alternative techniques to simplify the suturing process have been thought of. [19, 48]. Some laparocopic surgeons use omentopexy alone [18, 47]. Suture less techniques have been tried, in which fibrin glue alone or a gelatin sponge has been glued into the ulcer [18]. The downside of this technique is that is only can be used to close small perforations. To overcome this problem a biodegradable patch, that can be cut into any desirable size, has been tested in rats, with good results [50]. The combined laparoscopic-endoscopic repair also has been described as well [51].

POSTOPERATIVE MANAGEMENT

Reviewing literature all patients receive nasogastric draining for at least 48 hrs [16]. This however seems to be more “common practice” than evidence based medicine [49]. A routine nasogastric decompression does not achieve any of

its attended goals, should not applied to all and only be applied in selected cases, which has been supported by other trials as well [51-54].

This also means that oral feeding can be started early, as in colorectal surgery and that waiting for three days, as often is done according to protocol, is unnecessary [54-55].

Table 2 Post operative complications

Complication	Incidence
Pneumonia	3.6-30%
Wound infection	10-17%
Urinary tract infection	1.4-15%
Suture leak	2-16%
Abscess formation	0-9%
Heart problems (myocardial infarction, heart failure)	5%
Ileus	2-4%
Fistula	0.5-4%
Wound dehiscence	2.5-6%
Biliary leak	4.9%
Bleeding	0.6%
Re-operation	2-9%
Sepsis	2.5%
Stroke	4%
Death	5-11%

As can be seen in Table 2 wound infections represent the second most common complication after surgery for PPU. Also the incidence of sepsis is 2.5%. Preoperative intravenous administration of antibiotics has proven to lower the overall infection rate [56]. Although for most surgical procedures a single dose seems to be sufficient, in case of H. pylori infection triple therapy is recommended consisting of a proton pump inhibitor combined with clarithromycin and amoxicillin for 14 days [22, 33, 55, 56]. Upper gastrointestinal endoscopy is suggested to be performed after eight weeks to asses healing of the ulcers and to evaluate H.pylori status [55].

POSTOPERATIVE COMPLICATIONS.

The postoperative complication most common observed was pneumonia, followed by wound infection. An overview of all complications and their incidences, based on reviewing literature are listed in table 2 [19, 22, 25, 26, 48, 49, 56-61].

RISK FACTORS INFLUENCING OUTCOME

Mortality after surgery for perforated peptic ulcer is between 6-10% [26]. There are four main factors which can increase this mortality rate even up to 100%. These are

- ✓ Age > 60 years,
- ✓ Delayed treatment (>24hrs),

- ✓ Shock at time of the admission (systolic BP < 100 mmHg) and
- ✓ Comorbid diseases [25, 27].

Also gastric ulcers are associated with a two- to threefold increased mortality risk [25, 27]. Boey's score, based on scoring factors as shock on admission, confounding medical illness, and prolonged perforation, has been found to be a useful tool in predicting outcome [22, 29, 45, 57]. In the elderly mortality rate after surgery for PPU is three to five times higher and up to 50% [61]. This can be explained by the occurrence of co morbid medical diseases and delay > 24 hrs due to difficulties making the right diagnosis. In case of a perforated gastric ulcer or recurrent PUD (hemi)gastrectomy with vagotomy might be indicated, but overall simple closure is a safe procedure and there seem to be no need for definitive surgery in this group of patients since ulcer recurrence is only 14% [18, 61, 62].

MATERIALS AND METHODS

This study place was Department of General surgery, Rajivgandhi Government General Hospital attached to Madras Medical College, Chennai during the period of January 2013 to October 2013

The diagnosis of ulcer perforation was established by the admitting surgeon based on presenting complaints, clinical features and supported by radiological investigation and confirmed preoperatively. All patients of Duodenum part 1, Antrum, Pylorus and Prepyloric ulcer perforation are included in this study.

Patient factors Age, Sex, Clinical presentation including Pain site, Duration, Past peptic ulcer history and treatment, NSAID usage, Personal habits of smoking and consuming alcohol, Co morbid illnesses recorded.

Physical examination findings of Pulse rate, Respiratory rate, Blood pressure, Urine output monitored. Biochemical values of Urea, Creatinine, Electrolytes Sodium and Potassium, and Glucose level, Hematological values of Hemoglobin, Total count and Differential count noted. Duration between pain and surgery and admission and surgery were recorded.

ASA score, Laparotomy findings including site (Duodenum part 1,

Gastric antrum, Pylorus or Prepylorus), Size of perforation, Contamination volume and nature and procedure, Surgery duration recorded.

Post operative ventilator support, Cardiac support and Complications including Renal, Respiratory and Wound complications are followed and recorded.

Finally outcome of the surgery whether Death or Discharge and Total duration of hospital stay have been analyzed. Mortality means death following surgery within 28 days and morbidity means prolonged hospital stay and complications.

EXCLUSION CRITERIA

Cases of traumatic perforation

Cases of gastric body and duodenum part 2 perforations

Cases of iatrogenic perforations

TOOLS USED

The data were recorded in MS-excel and were analyzed using Statistical Package for Social Science (SPSS-16). The methods like Frequency analysis, Cross tabulation, Univariate and Multivariate analysis have been employed.

Following are the analysis of the study.

AGE GROUP

Age	FINAL OUTCOME	Morbidity (hospital stay) <= 7 days	8-14 days	15-21 days	22-28 days	>= 29 days	Total
Less than 20	Discharge		4				4
	% of Total		100.0%				100.0%
20-39	Death	4	0	0	0	0	4
	% of Total	11.8%	.0%	.0%	.0%	.0%	11.8%
	Discharge	3	21	3	2	1	30
	% of Total	8.8%	61.8%	8.8%	5.9%	2.9%	88.2%
40-59	Death	8	0	0			8
	% of Total	22.2%	.0%	.0%			22.2%
	Discharge	4	21	3			28
	% of Total	11.1%	58.3%	8.3%			77.8%
60 and above	Death	10	2	0			12
	% of Total	38.5%	7.7%	.0%			46.2%
	Discharge	0	13	1			14
	% of Total	.0%	50.0%	3.8%			53.8%

The above table shows the frequency of the patients age distribution. It is inferred that the patients whose age group is 60 and above are subject to death at large extent (46.2 percent) and their hospital stay is <=7 days due to death.

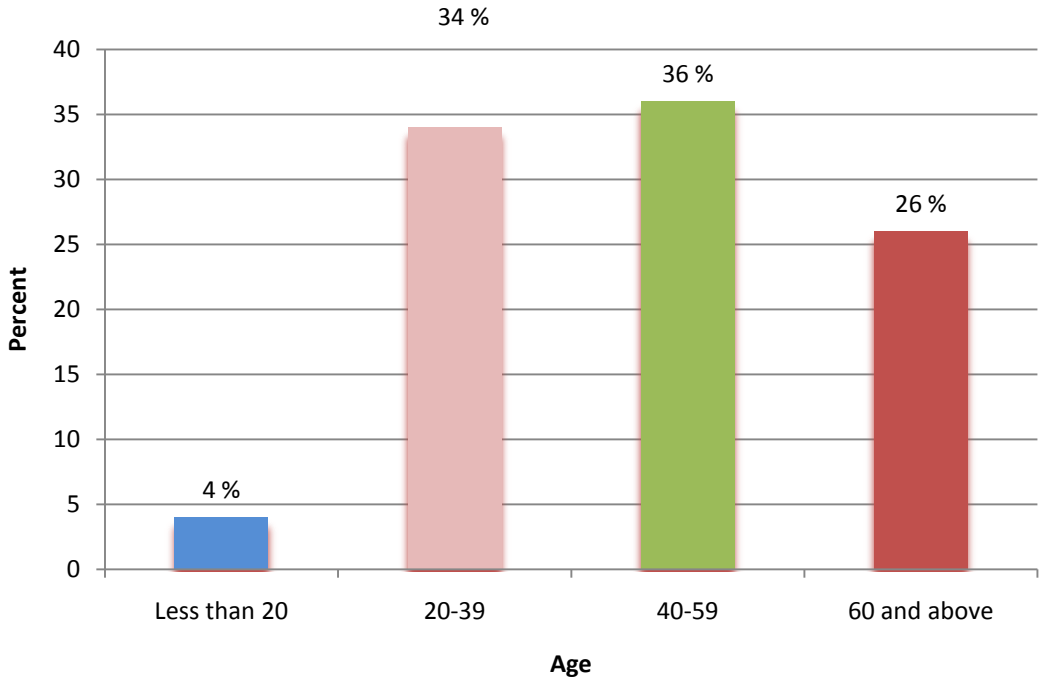
The following table shows the Multivariate analysis to test the significance of Age on the Morbidity and Mortality of the patient. It is found that Age has significant effect on the Morbidity and Mortality of the patient as their

significant value is less than 5%. The size of the effect is 75.7 % on Morbidity and 10.3 % on Mortality.

Multivariate analysis

Source	Dependent Variable	Type III Sum of Squares	Df	Mean Square	F	*Sig.*	Partial Eta Squared
Age	Morbidity	90.986	1	90.986	143.123	*.000*	.757
	Mortality	.929	1	.929	5.272	*.026*	.103

AGE DISTRIBUTION

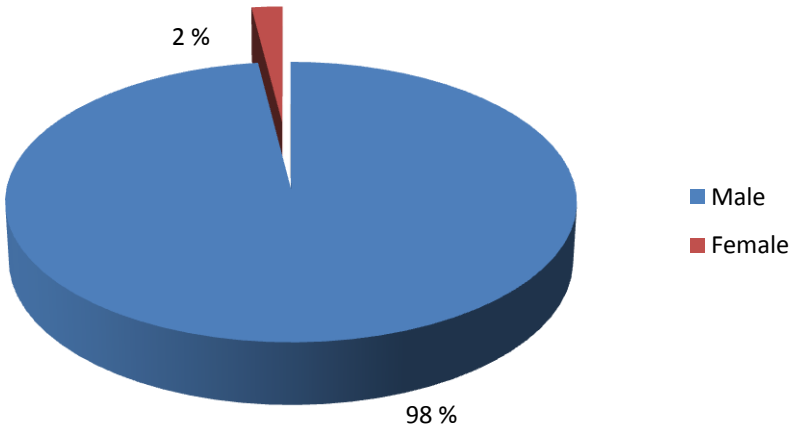


SEX

Sex	Mortality		Morbidity					Total
			<=7 days	8-14 days	15-21 days	22-28 days	>=29 days	
Male	Death	Count	20	2	0	0	0	22
		% of Total	20.4%	2.0%	.0%	.0%	.0%	22.4%
	Discharge	Count	7	59	7	2	1	76
		% of Total	7.1%	60.2%	7.1%	2.0%	1.0%	77.6%
Female	Death	Count	2					2
		% of Total	100.0%					100.0%

The number of Male and Female patients under the study are 98 and 2 respectively are noted.

SEX

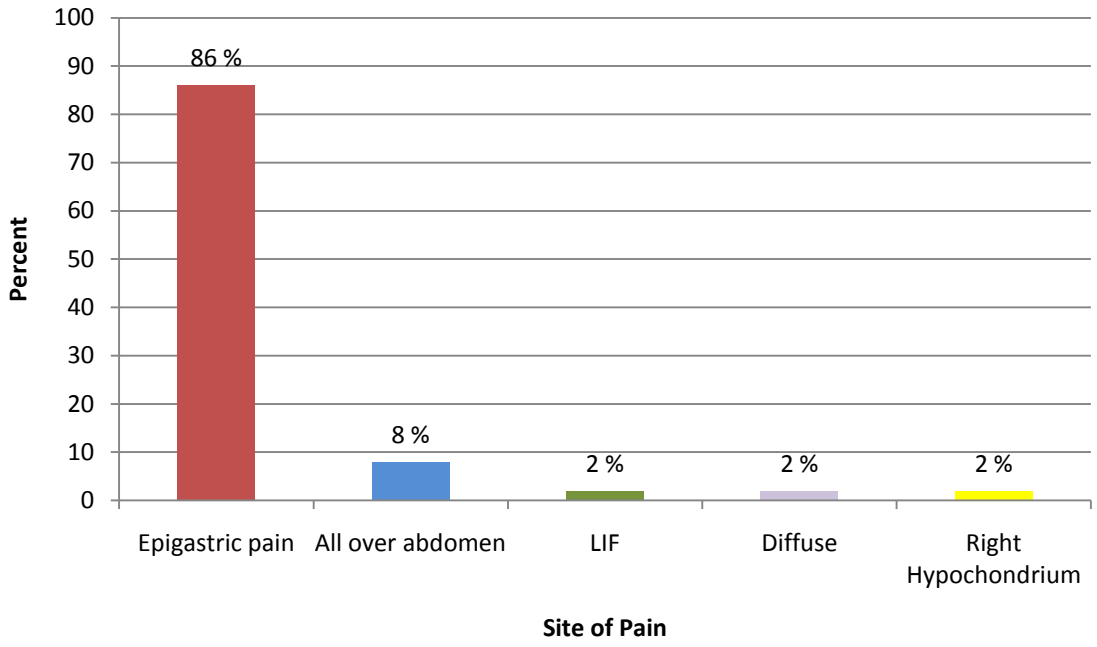


SITE OF PAIN

Site of Pain	No. of patients	Percentage
Epigastric pain	86	86.0
All over abdomen	8	8.0
LIF	2	2.0
Diffuse	2	2.0
Right Hypochondrium	2	2.0
Total	***100***	***100.0***

It is inferred from the above table that 86 % of the Patients had Epigastric pain and the remaining 12 % did not have the same.

SITE OF PAIN



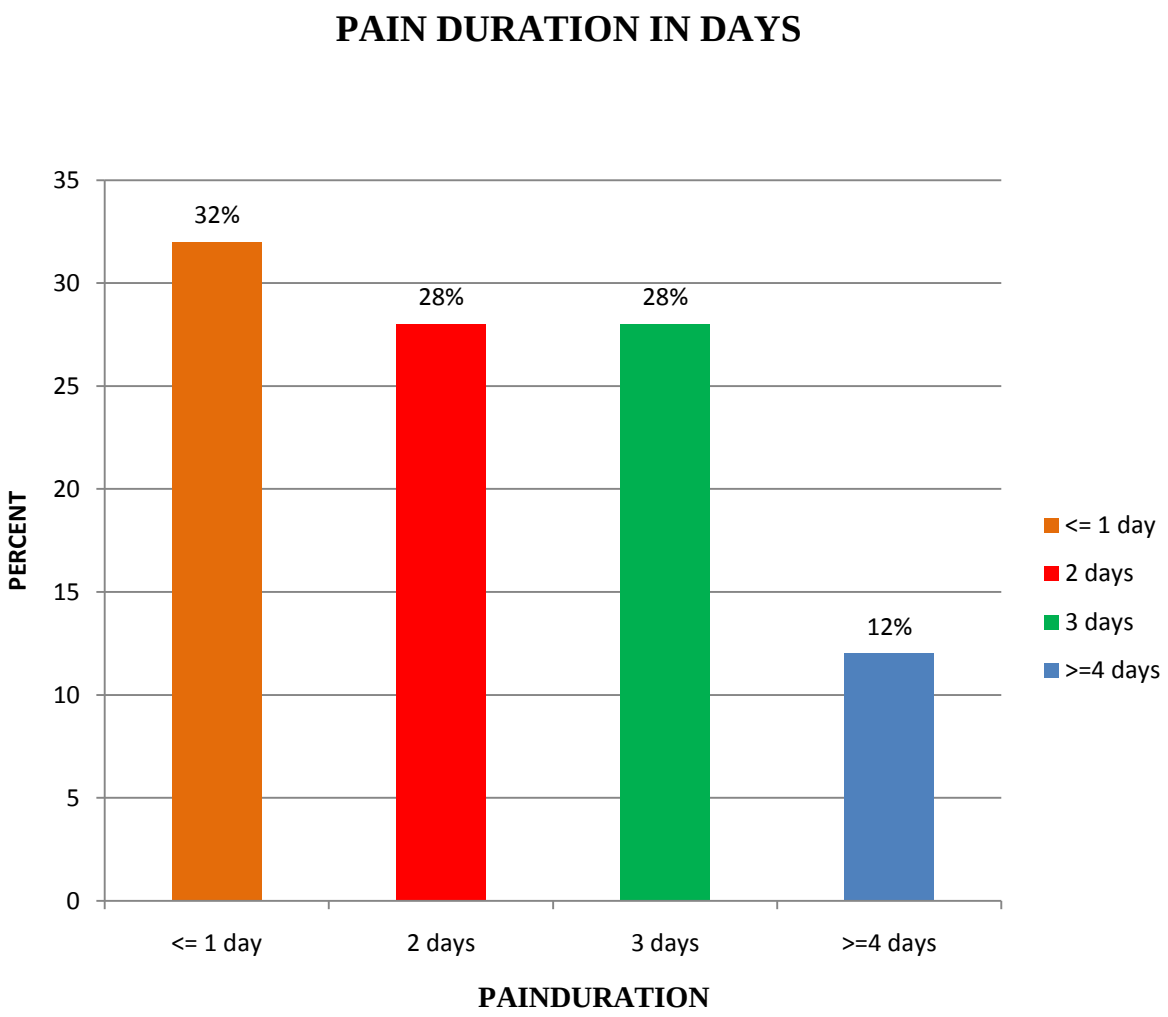
PAIN DURATION

Painduration	Mortality		Morbidity					Total
			<= 7 days	8-14 days	15-21 days	22-28 days	>= 29 days	
<= 1 day	Death	Count	2	2				4
		% of Total	6.2%	6.2%				12.5%
	Discharge	Count	2	26				28
		% of Total	6.2%	81.2%				87.5%
2 days	Death	Count	4	0	0	0	0	4
		% of Total	14.3%	.0%	.0%	.0%	.0%	14.3%
	Discharge	Count	2	17	3	1	1	24
		% of Total	7.1%	60.7%	10.7%	3.6%	3.6%	85.7%
3 days	Death	Count	10	0	0	0		10
		% of Total	35.7%	.0%	.0%	.0%		35.7%
	Discharge	Count	3	11	3	1		18
		% of Total	10.7%	39.3%	10.7%	3.6%		64.3%
>=4 days	Death	Count	6	0	0			6
		% of Total	50.0%	.0%	.0%			50.0%
	Discharge	Count	0	5	1			6
		% of Total	.0%	41.7%	8.3%			50.0%

The above table shows the cross tabulation of Pain duration, Morbidity and Mortality. It is observed from the following table that Pain duration in days has significant effect on the Mortality of the patient. And the size of the effect is 10.3 percent. Whereas the Pain duration in days does not has any effect on the Morbidity of the patient.

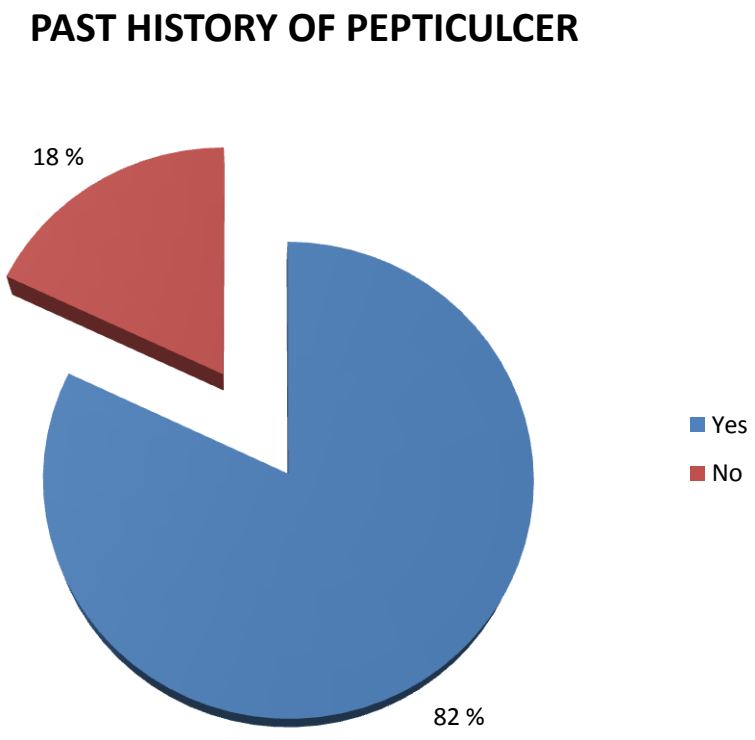
Multivariate analysis

Source	Dependent Variable	Type III Sum of Squares	Df	Mean Square	F	*Sig.*	Partial Eta Squared
Pain duration	Mortality	1.883	3	.628	3.683	*.015*	.103
	Morbidity	2.762	3	.921	1.842	*.145*	.054



PAST HISTORY OF PEPTICULCER

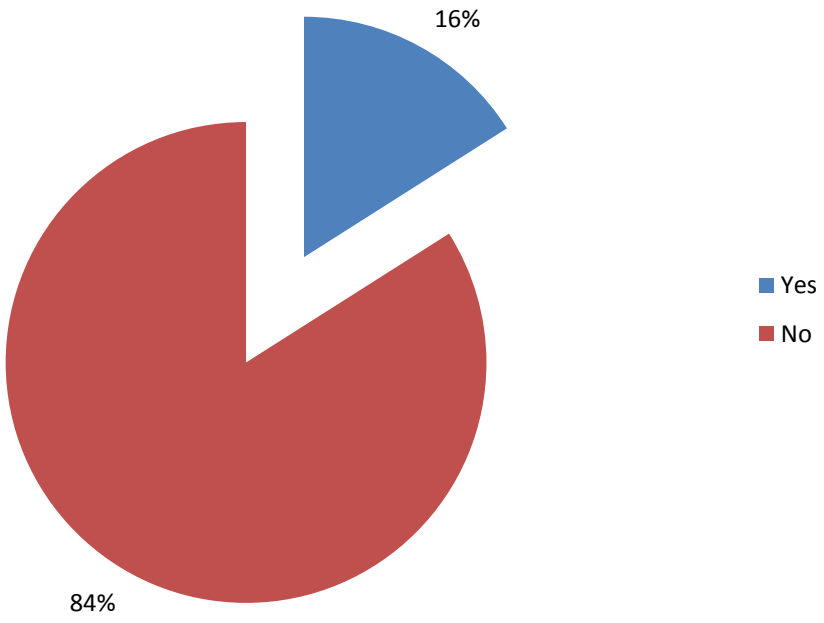
Past history of Pepticulcer	No. of patients	Percent
Yes	82	82.0
No	18	18.0
Total	**100**	**100.0**



NSAID USAGE

History of NSAID	No. of patients	Percent
Yes	16	16.0 %
No	84	84.0 %
Total	**100**	**100.0**

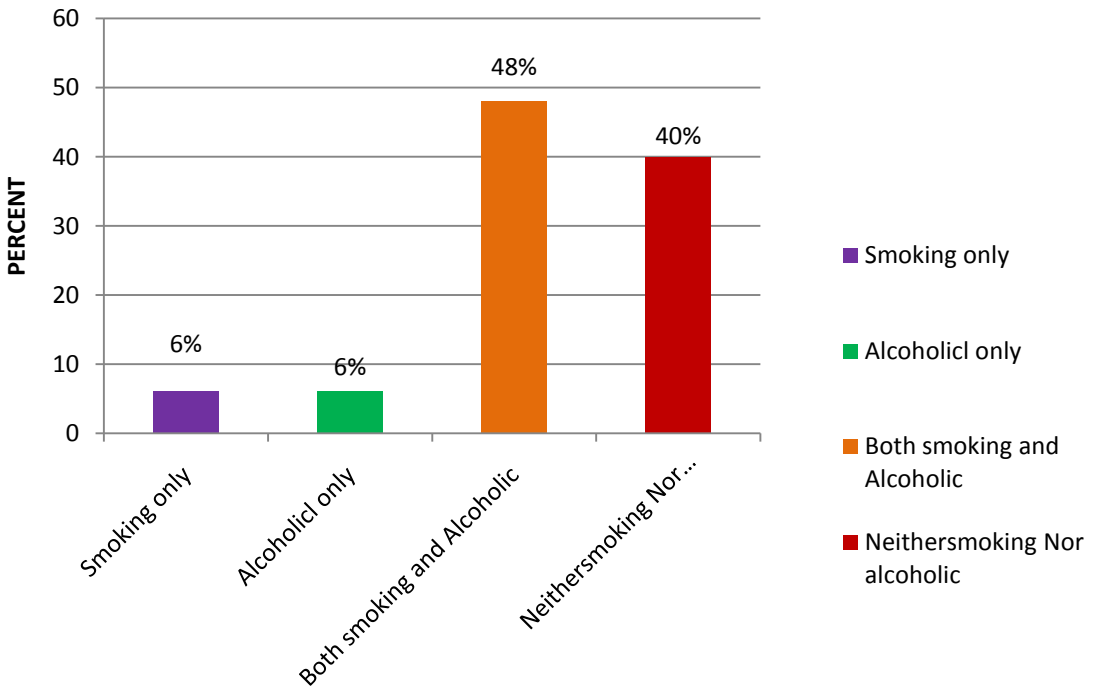
NSAID USAGE



SMOKING AND ALCOHOL HABITS

Smoking and Alcohol habits	No. of patients	Percent
Smoking only	6	6.0 %
Consumes alcohol only	6	6.0 %
Both smoking and Alcohol	48	48.0 %
Neither smoking Nor alcohol	40	40.0 %
Total	**100**	**100.0**

SMOKING AND ALCOHOL HABITS

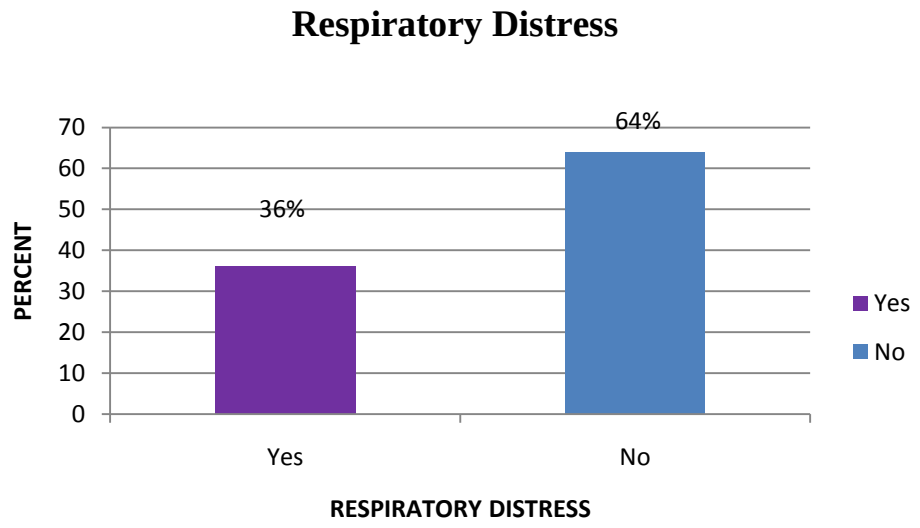


COMORBID ILLNESS

Comorbid illness		No. of patients	Percent
YES	BA	4	4.0 %
	DCLD	2	2.0 %
	DM,CRF	2	2.0 %
	HIV,Hep B	2	2.0 %
	PVD,BA	2	2.0 %
NO		88	88.0 %
Total		**100**	**100.0**

RESPIRATORY DISTRESS (TACHYPNEA)

Respiratory Distress	No. of Patients	Percent
Yes	36	36.0 %
No	64	64.0 %
Total	**100**	**100.0**

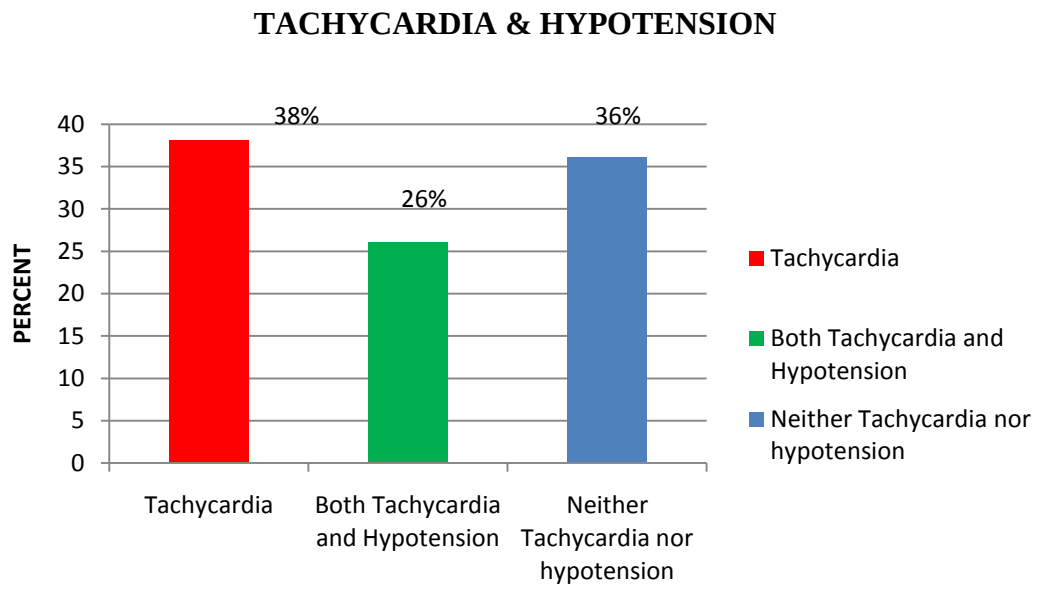


ANEMIA

Anemia	Frequency	Percent
Yes	20	20.0
No	80	80.0
Total	**100**	**100.0**

TACHYCARDIA & HYPOTENSION

Tachycardia Hypotension	No. of Patients	Percent
Tachycardia	38	38.0 %
Both Tachycardia and Hypotension	26	26.0 %
Neither Tachycardia nor hypotension	36	36.0 %
Total	**100**	**100.0**



SHOCK

Shock	Mortality		Morbidity					
			<=7 days	8-14 days	15-21 days	22-28 days	>= 29 days	**Total**
Class I	Death	Count	0	2	0	0		2
		% of Total	.0%	4.5%	.0%	.0%		4.5%
	Discharge	Count	7	32	2	1		42
		% of Total	15.9%	72.7%	4.5%	2.3%		95.5%
Class II	Death	Count	6	0	0	0	0	6
		% of Total	20.0%	.0%	.0%	.0%	.0%	20.0%
	Discharge	Count	0	18	4	1	1	24
		% of Total	.0%	60.0%	13.3%	3.3%	3.3%	80.0%
Class III	Death	Count	16	0	0			16
		% of Total	/61.5%	.0%	.0%			61.5%
	Discharge	Count	0	9	1			10
		% of Total	.0%	34.6%	3.8%			38.5%

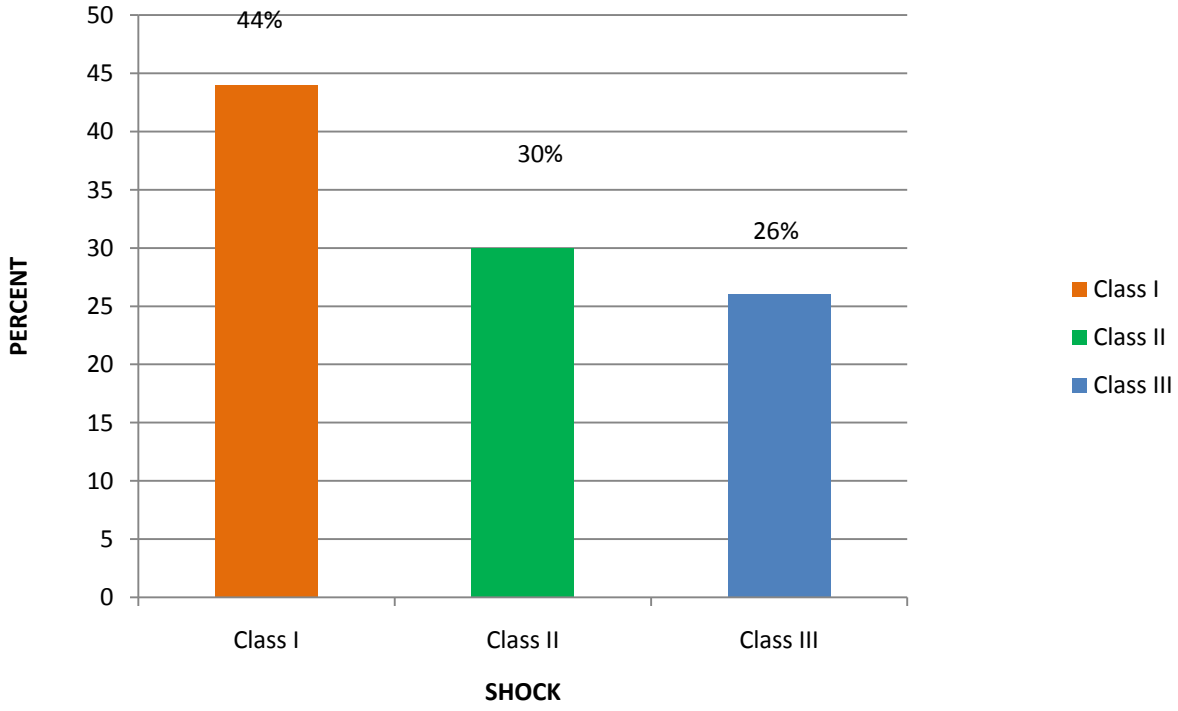
The above table shows the cross tabulation of Shock, Mortality and Morbidity of the patients under the purview of the study. Death is more in the patients who had class III shock.

The Multivariate analysis has been done to test the significance of Shock on the Mortality and Morbidity of the patient. It is observed from the following table that the factor, Shock has significant effect on both the Mortality and Morbidity of the patient. The shock contributes 29.5 percent and 13.6 percent to the Mortality and Morbidity of the patient respectively.

Multivariate analysis

Source	Dependent Variable	Type III Sum of Squares	df	Mean Square	F	*Sig.*	Partial Eta Squared
Shock	Mortality	5.377	2	2.689	20.274	*.000*	.295
	Morbidity	6.908	2	3.454	7.642	*.001*	.136

SHOCK



PREOPERATIVE LRI

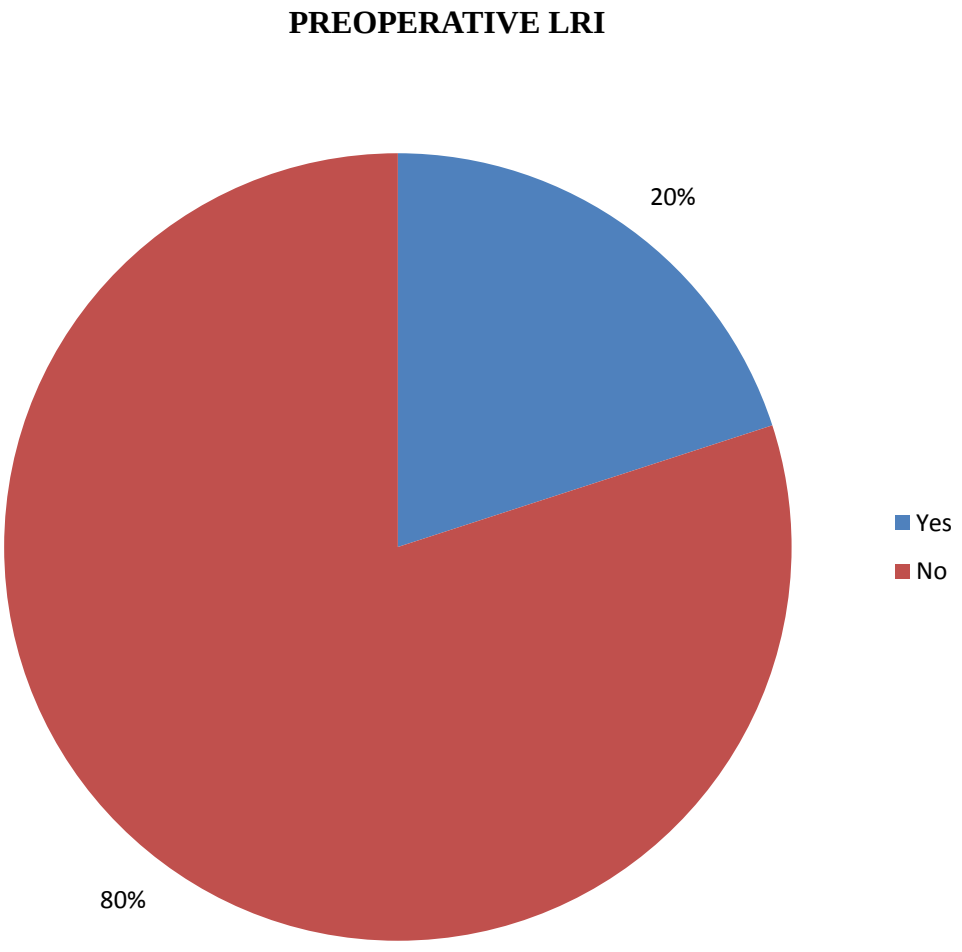
Preoperative LRI			Morbidity					Total
Mortality			<= 7 days	8-14 days	15-21 days	22-28 days	>= 29 days	
Yes	Death	Count	8	0	0			8
		% of Total	40.0%	.0%	.0%			40.0%
	Discharge	Count	0	11	1			12
		% of Total	.0%	55.0%	5.0%			60.0%
No	Death	Count	14	2	0	0	0	16
		% of Total	17.5%	2.5%	.0%	.0%	.0%	20.0%
	Discharge	Count	7	48	6	2	1	64
		% of Total	8.8%	60.0%	7.5%	2.5%	1.2%	80.0%

The cross tabulation of Preoperative LRI, Morbidity and Mobility of the patients under the study has been presented in the above table. The preoperative LRI is related to 40% of death.

To test the significance of Preoperative LRI on the morbidity and Mortality of the patient, the Multivariate analysis has been adopted and presented in the following table. The result exhibit that the Preoperative LRI has significant effect on the Mortality of the patients as the significant values is less than 5 %. The Preoperative LRI contributes 33.5 percent to the Mortality. The preoperative LRI does not have any significant effect on the Morbidity.

Multivariate analysis

Source	Dependent Variable	Type III Sum of Squares	df	Mean Square	F	*Sig.*	Partial Eta Squared
Preoperative LRI	Mortality	.640	1	.640	3.564	*.042*	.335
	Morbidity	1.000	1	1.000	1.970	*.164*	.020



CXR AIR UNDER DIAPHARGM

CXR AIR UNDER DIAPHARGM	No. of Patients	Percent
Yes	76	76.0 %
No	24	24.0 %
Total	**100**	**100.0**

BLOOD GLUCOSE

Blood Glucose	No. of Patients	Percent
<60	2	2.0
60-120	82	82.0
121-140	14	14.0
>140	2	2.0
Total	**100**	**100.0**

UREA

Urea	No. of Patients	Percent
Normal (<=30)	**38**	**38.0**
High (>30)	**62**	**62.0**
Total	**100**	**100.0**

CREATININE

Creatinine	No. of Patients	Percent
Normal	54	54.0
High	46	46.0
Total	**100**	**100.0**

SODIUM

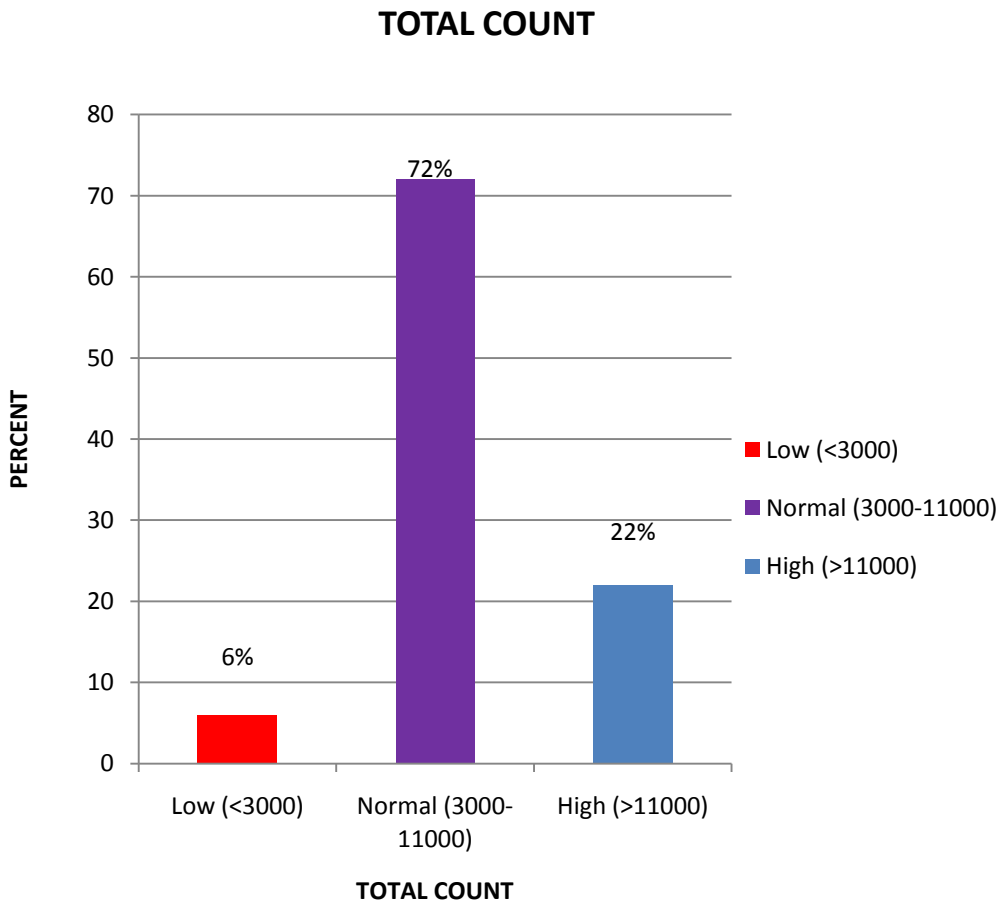
Sodium	No. of Patients	Percent
Low (<136)	20	20.0
Normal (136-146)	80	80.0
Total	**100**	**100.0**

POTTASSIUM

Pottassium	No. of Patients	Percent
Normal (3.5-5)	74	74.0
High (>5)	12	12.0
Total	**100**	**100.0**

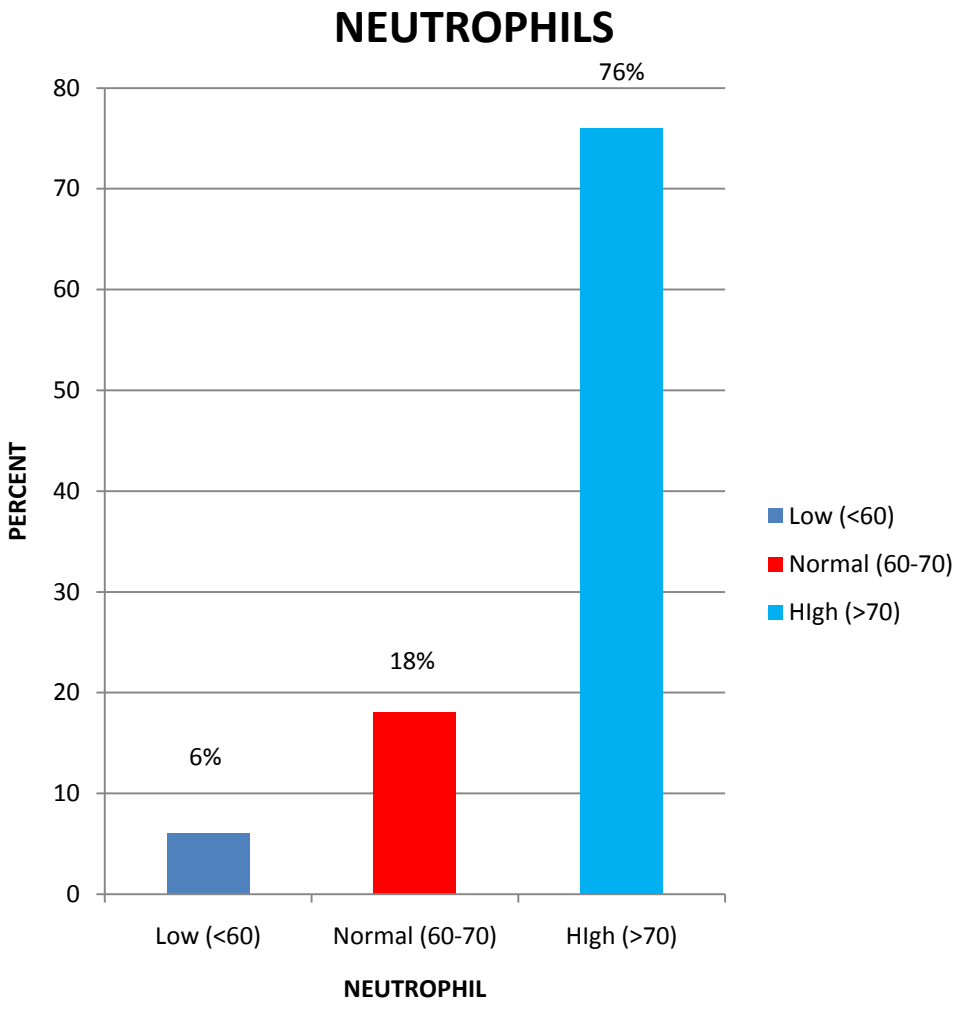
TOTAL COUNT

Total count	No. of Patients	Percent
Low (<3000)	6	6.0
Normal (3000-11000)	72	72.0
High (>11000)	22	22.0
Total	**100**	**100.0**



NEUTROPHILS

Neutrophils	No. of Patients	Percent
Low (<60%)	6	6.0
Normal (60-70%)	18	18.0
HIgh (>70%)	76	76.0
Total	**100**	**100.0**



LYMPHOCYTES

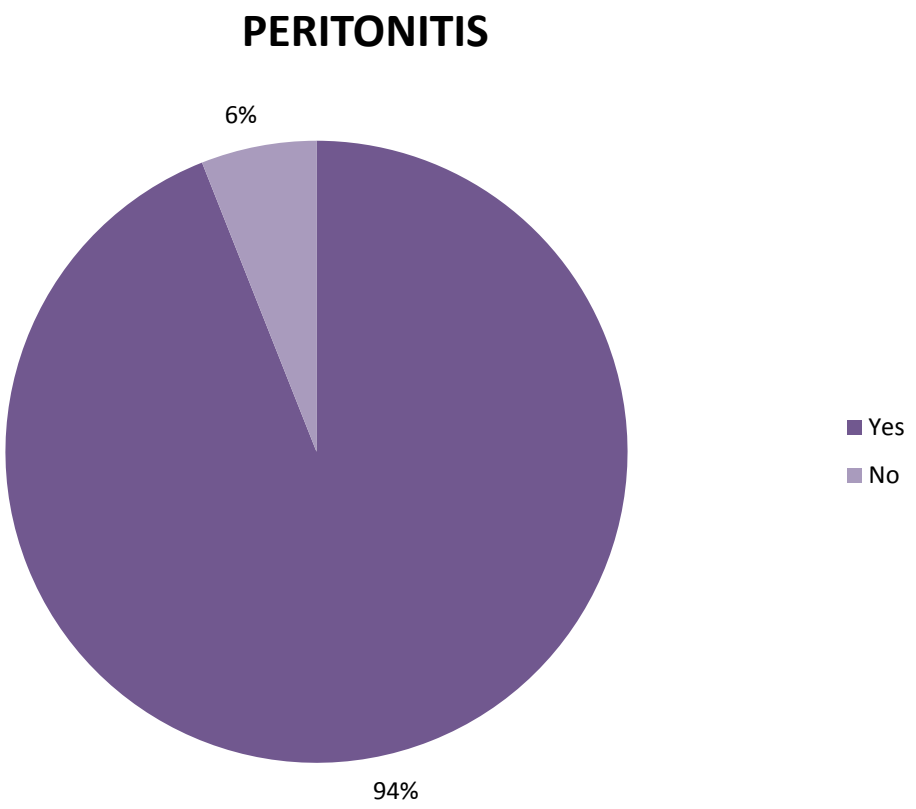
Lymphocytes	No. of Patients	Percent
Low (<20%)	66	66.0
Normal (20-30%)	22	22.0
High (>30%)	12	12.0
Total	**100**	**100.0**

HEAMOGLOBIN

Heamoglobin	No. of Patients	Percent
Low (<12)	56	56.0
Normal (12-16)	44	44.0
Total	**100**	**100.0**

PERITONITIS

Peritonitis	No. of patients	Percent
Yes	94	94.0
No	6	6.0
Total	**100**	**100.0**



ASA SCORE

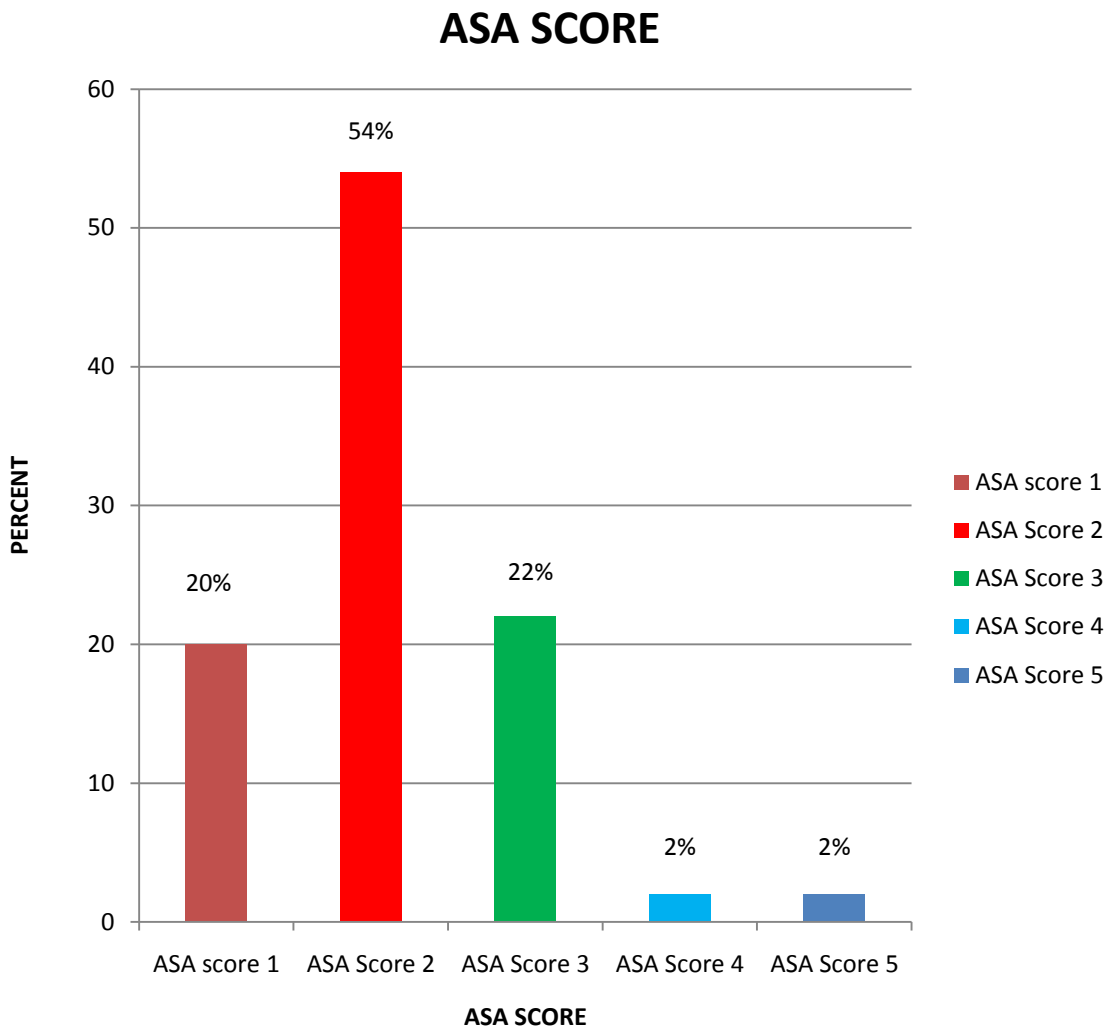
ASAscore	Mortality		Morbidity <= 7 days	8-14 days	15-21 days	22-28 days	>= 29 days	Total
ASA score 1	Discharge	Count	2	14	2	1	1	20
		% of Total	10.0%	70.0 %	10.0%	5.0%	5.0%	100.0 %
ASA Score 2	Death	Count	4	2	0	0		6
		% of Total	7.4%	3.7%	.0%	.0%		11.1%
	Discharge	Count	5	38	4	1		48
		% of Total	9.3%	70.4 %	7.4%	1.9%		88.9%
ASA Score 3	Death	Count	14	0	0			14
		% of Total	63.6%	.0%	.0%			63.6%
	Discharge	Count	0	7	1			8
		% of Total	.0%	31.8 %	4.5%			36.4%
ASA Score 4	Death	Count	2					2
		% of Total	100.0%					100.0 %
ASA Score 5	Death	Count	2					2
		% of Total	100.0%					100.0 %

The above table shows the cross tabulation of ASA Score, Mortality and Morbidity of the patients selected under the study. ASA score of 4 and 5 had 100% mortality and score of 3 had 64%.

The following table shows that the ASA score has significant effect on both the Morbidity and Mortality of the patient as their significance value is less than 5 % significance value. The effect of ASA score on the Mortality and Morbidity of the patient is 42.8 percent and 21.4 percent respectively.

Multivariate analysis

Source	Dependent Variable	Type III Sum of Squares	df	Mean Square	F	*Sig.*	Partial Eta Squared
ASA score	Mortality	7.816	4	1.954	17.807	*.000*	.428
	Morbidity	10.848	4	2.712	6.457	*.000*	.214



DURATION BETWEEN PAIN AND SURGERY

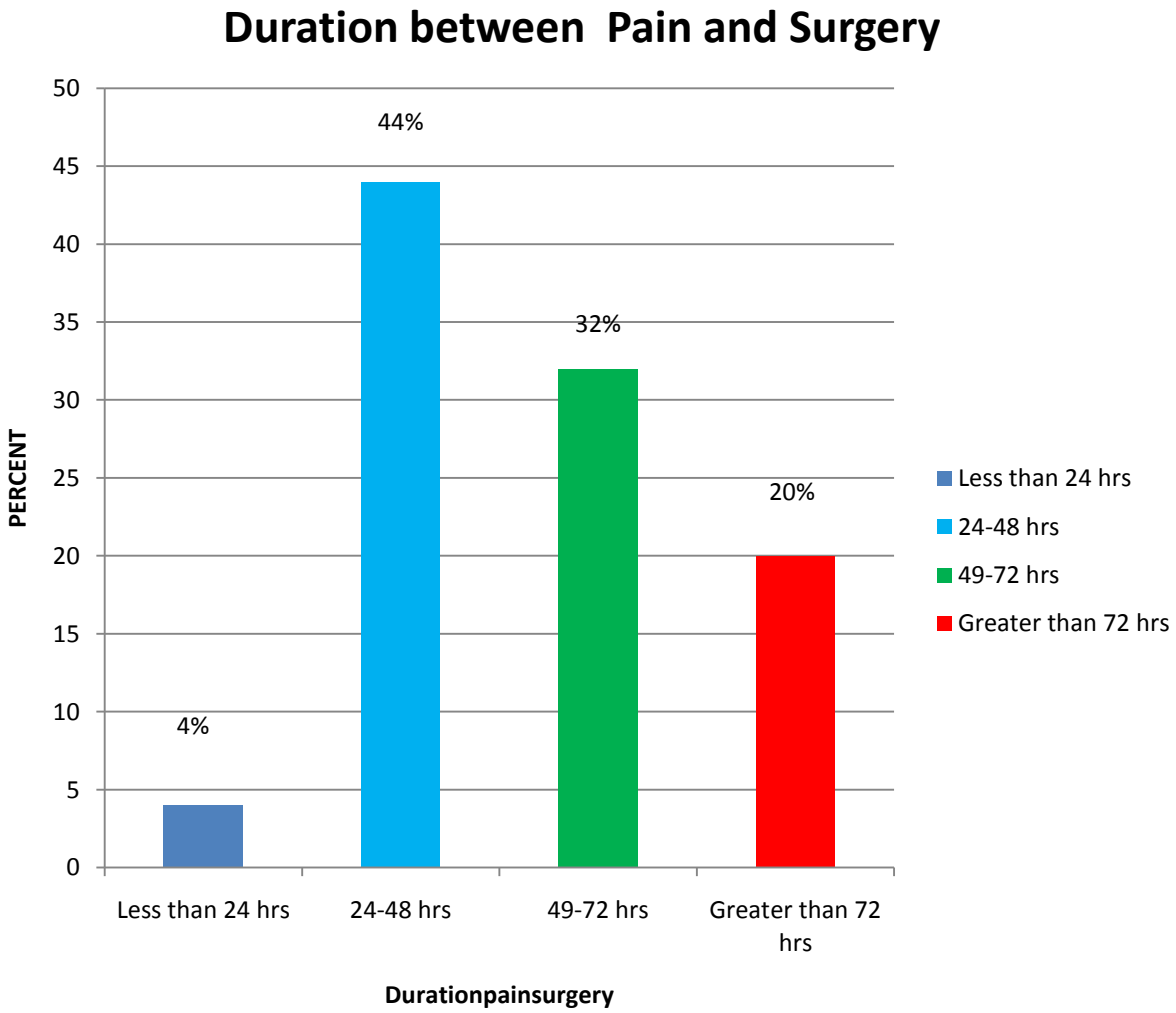
Duration- pain surgery	Mortality		Morbidity					Total
			<= 7 days	8-14 days	15-21 days	22-28 days	>= 29 days	
Less than 24 hrs	Discharge	Count		4				4
		% of Total		100.0%				100.0%
24-48 hrs	Death	Count	6	2	0			8
		% of Total	13.6%	4.5%	.0%			18.2%
	Discharge	Count	4	31	1			36
		% of Total	9.1%	70.5%	2.3%			81.8%
49-72 hrs	Death	Count	10	0	0	0	0	10
		% of Total	31.2%	.0%	.0%	.0%	.0%	31.2%
	Discharge	Count	3	14	3	1	1	22
		% of Total	9.4%	43.8%	9.4%	3.1%	3.1%	68.8%
Greater than 72 hrs	Death	Count	6	0	0	0		6
		% of Total	30.0%	.0%	.0%	.0%		30.0%
	Discharge	Count	0	10	3	1		14
		% of Total	.0%	50.0%	15.0%	5.0%		70.0%

The above table shows the cross tabulation between the Duration between pain and surgery, Morbidity and Mortality. Duration of more than 48 hrs between pain and surgery has significant mortality.

It is inferred from the following table that the duration between pain and surgery has significant effect on the Mortality and Morbidity of the patient, as their significance values is greater than 5 %.

Multivariate analysis

Source	Dependent Variable	Type III Sum of Squares	df	Mean Square	F	*Sig.*	Partial Eta Squared
Duration pain surgery	Mortality	.620	3	.207	1.125	*.043*	.034
	Morbidity	.422	3	.141	.268	*.048*	.008



DURATION BETWEEN ADMISSION AND SURGERY

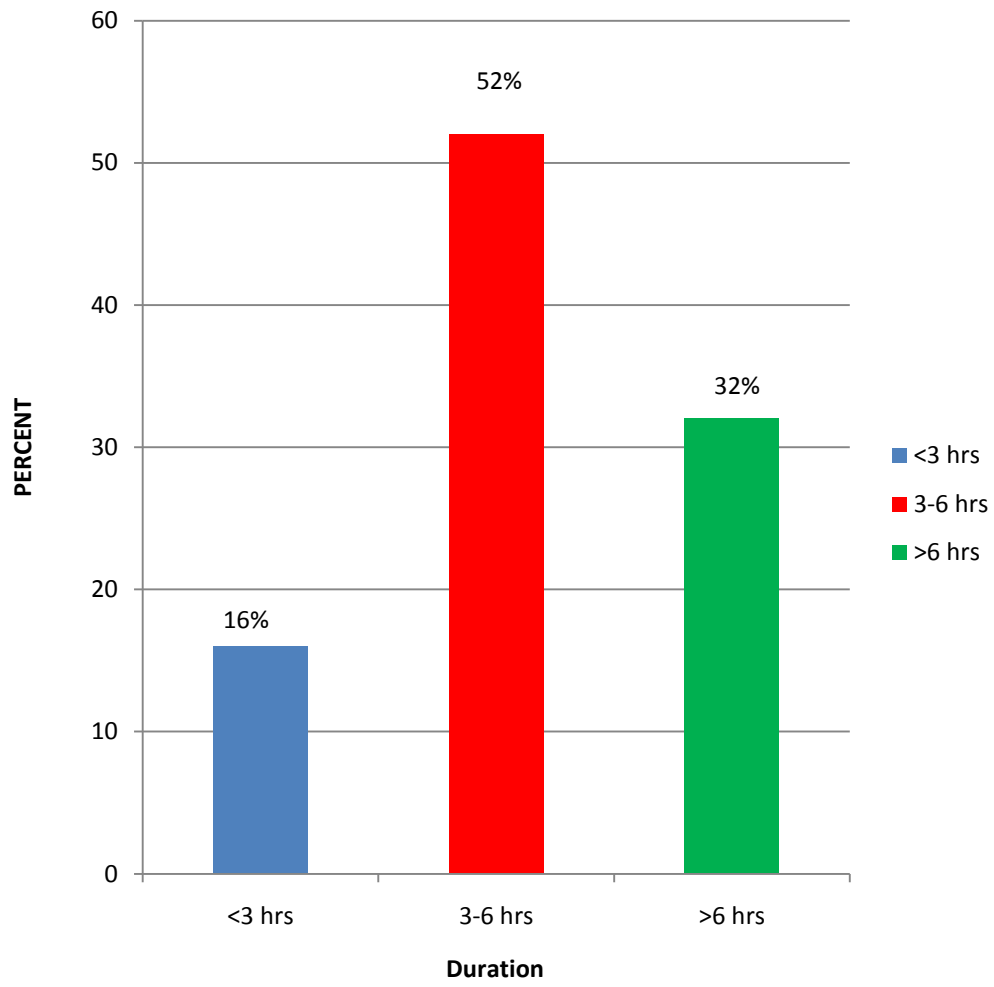
Duration Admn Surgery	Mortality		Morbidity					Total
			<= 7 days	8-14 days	15-21 days	22-28 days	>= 29 days	
<3 hrs	Death	Count	8	0				8
		% of Total	50.0%	.0%				50.0%
	Discharge	Count	1	7				8
		% of Total	6.2%	43.8%				50.0%
3-6 hrs	Death	Count	10	0	0	0	0	10
		% of Total	19.2%	.0%	.0%	.0%	.0%	19.2%
	Discharge	Count	4	30	5	2	1	42
		% of Total	7.7%	57.7%	9.6%	3.8%	1.9%	80.8%
>6 hrs	Death	Count	4	2	0			6
		% of Total	12.5%	6.2%	.0%			18.8%
	Discharge	Count	2	22	2			26
		% of Total	6.2%	68.8%	6.2%			81.2%

Cross tabulation between the admission surgery duration, Mortality and Morbidity of the patient has been exhibited in the above table. To test the significance of Admission surgery duration on the mortality and morbidity of the patient, Multivariate analysis has been analyzed. The result has been exhibited in the following table. It is inferred that the duration between admission & surgery has a significant effect on the Mortality and Morbidity. The duration between admission and surgery has 17.1 % and 6.7% effect on the Mortality and Morbidity respectively.

Multivariate analysis

Source	Dependent Variable	Type III Sum of Squares	Df	Mean Square	F	*Sig.*	Partial Eta Squared
DurationAdmn Surgery	Mortality	1.288	2	.644	3.685	*.029*	.171
	Morbidity	3.389	2	1.695	3.471	*.035*	.067

Duration between Admission and Surgery



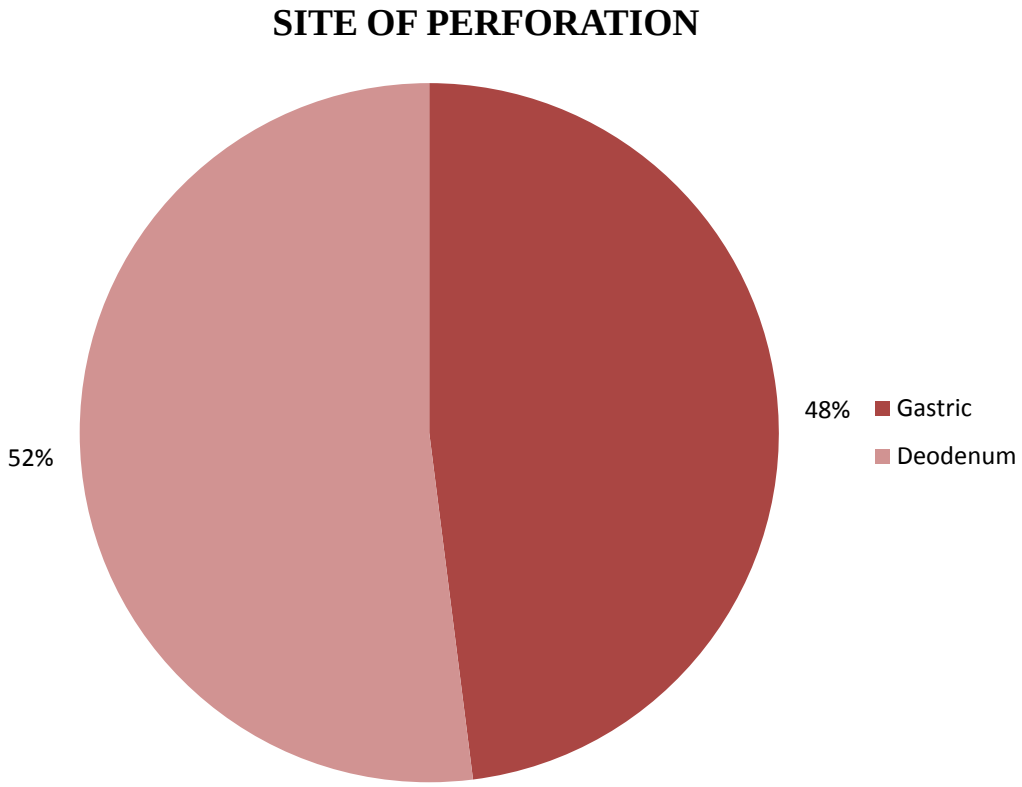
SITE OF PERFORATION

Site of Perforation	Mortality		Morbidity					Total
			<= 7 days	8-14 days	15-21 days	22-28 days	>= 29	
Gastric	Death	Count	14	2	0		0	16
		% of Total	29.2%	4.2%	.0%		.0%	33.3%
	Discharge	Count	1	26	4		1	32
		% of Total	2.1%	54.2%	8.3%		2.1%	66.7%
Duodenum	Death	Count	8	0	0	0		8
		% of Total	15.4%	.0%	.0%	.0%		15.4%
	Discharge	Count	6	33	3	2		44
		% of Total	11.5%	63.5%	5.8%	3.8%		84.6%

The cross tabulation of frequency of Site of perforation, Morbidity and Mortality has been presented in the above table. The significance of Site of perforation to the Morbidity and Mobility has been exhibited in the below table. The result shows that the site of perforation is significant to the Mortality as the significance value is less than 5 %. On the other hand site of perforation is not significant to the Morbidity (Sig, 0.824 is > than .05). The size of effect of perforation is significant to the extent of 14.4 %.

Multivariate analysis

Source	Dependent Variable	Type III Sum of Squares	Df	Mean Square	F	*Sig.*	Partial Eta Squared
Site of Perforation	Mortality	.804	1	.804	4.520	*.036*	.144
	Morbidity	.026	1	.026	.050	*.824*	.001



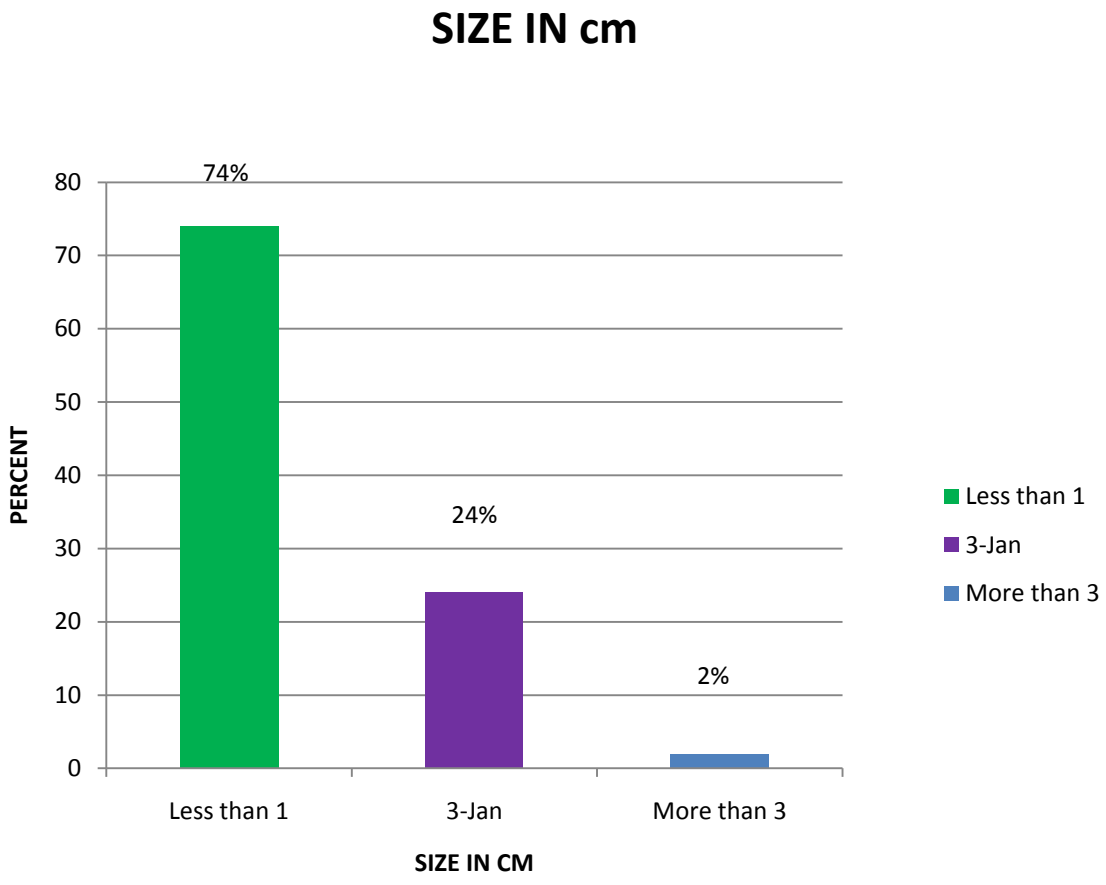
SIZE

Sizeincm	Mortality		Morbidity <= 7 days	8-14 days	15-21 days	22-28 days	>= 29 days	Total
Less than 1	Death	Count	10	2	0	0	0	12
		% of Total	13.5%	2.7%	.0%	.0%	.0%	16.2%
	Discharge	Count	6	48	5	2	1	62
		% of Total	8.1%	64.9%	6.8%	2.7%	1.4%	83.8%
1-3	Death	Count	10	0	0			10
		% of Total	41.7%	.0%	.0%			41.7%
	Discharge	Count	1	11	2			14
		% of Total	4.2%	45.8%	8.3%			58.3%
More than 3	Death	Count	2					2
		% of Total	100.0%					100.0%

The frequency of the Size in cm, Morbidity and Mortality has been presented in the cross tabulation in the above table. It is observed in the following table that the size in cm has been significant on the Mortality to the extent of 12.9 percent and Morbidity has significant effect on the Morbidity to the extent of 6.6 percent. Hence Size in cm significant to the Mortality and Morbidity of the patient.

Multivariate analysis

Source	Dependent Variable	Type III Sum of Squares	df	Mean Square	F	*Sig.*	Partial Eta Squared
Size in cm	Mortality	2.353	2	1.176	7.182	*.001*	.129
	Morbidity	3.341	2	1.671	3.418	*.037*	.066



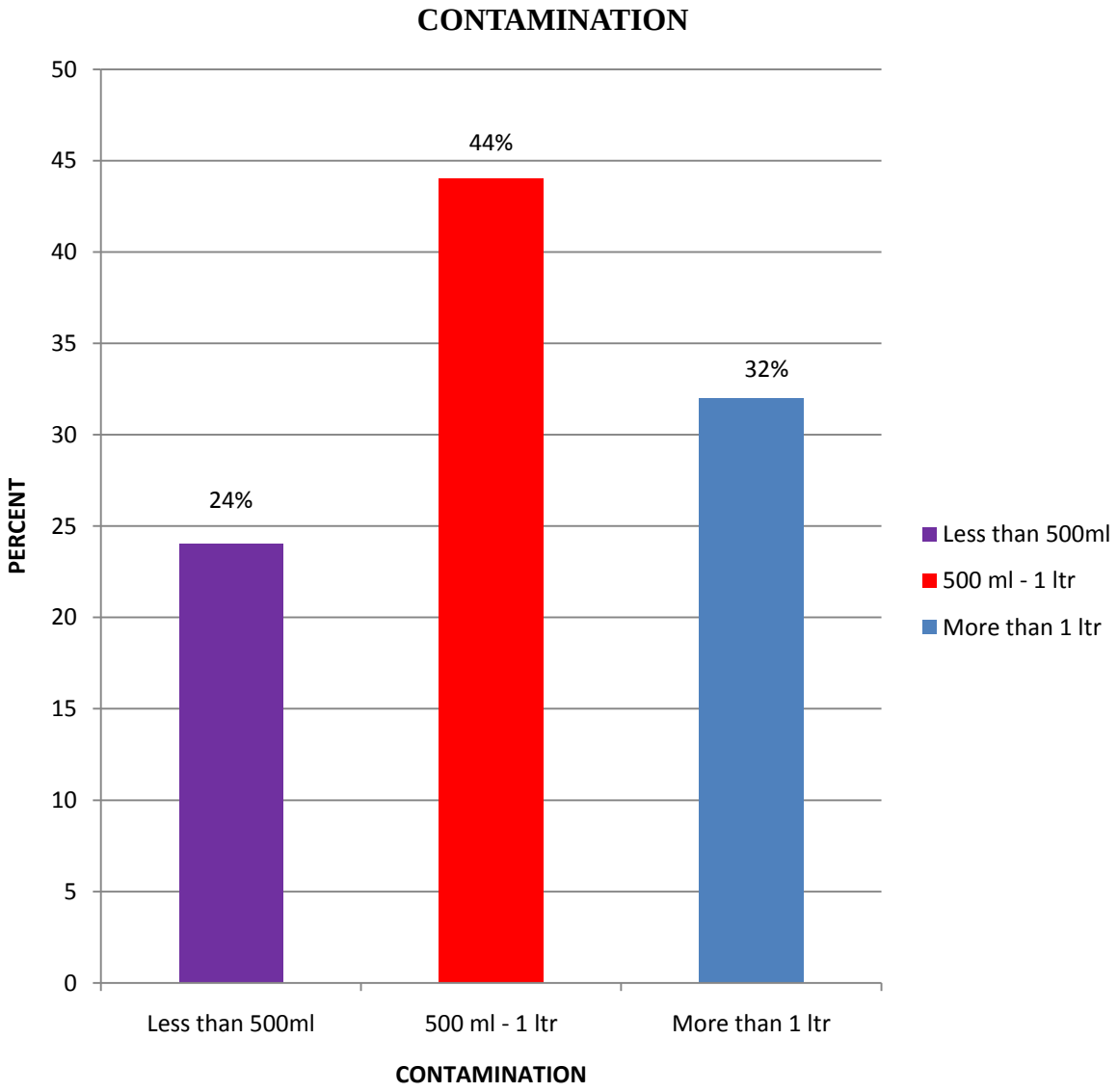
CONTAMINATION

Contamination	Mortality		Morbidity					Total
			<= 7 days	8-14 days	15-21 days	22-28 days	>= 29 days	
Less than 500ml	Death	Count	2	0	0			2
		% of Total	8.3%	.0%	.0%			8.3%
	Discharge	Count	2	19	1			22
		% of Total	8.3%	79.2%	4.2%			91.7%
500 ml - 1 ltr	Death	Count	6	2	0	0		8
		% of Total	13.6%	4.5%	.0%	.0%		18.2%
	Discharge	Count	4	28	3	1		36
		% of Total	9.1%	63.6%	6.8%	2.3%		81.8%
More than 1 ltr	Death	Count	14	0	0	0	0	14
		% of Total	43.8%	.0%	.0%	.0%	.0%	43.8%
	Discharge	Count	1	12	3	1	1	18
		% of Total	3.1%	37.5%	9.4%	3.1%	3.1%	56.2%

The cross tabulation of contamination, Morbidity and Mortality has been presented in the cross tabulation in the above table. The following table shows the Multivariate analysis to check if the Contamination has any significant effect on the Mortality and Morbidity of the patient. It results that the Contamination has significant contribution to the Mortality and Morbidity. It is significant to the extent of 10.9 % and 9.04 % to the Mortality and Morbidity respectively.

Multivariate analysis

Source	Dependent Variable	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Contamination	Mortality	1.986	2	.993	5.927	.004	.109
	Morbidity	.224	2	.112	.215	.007	.094



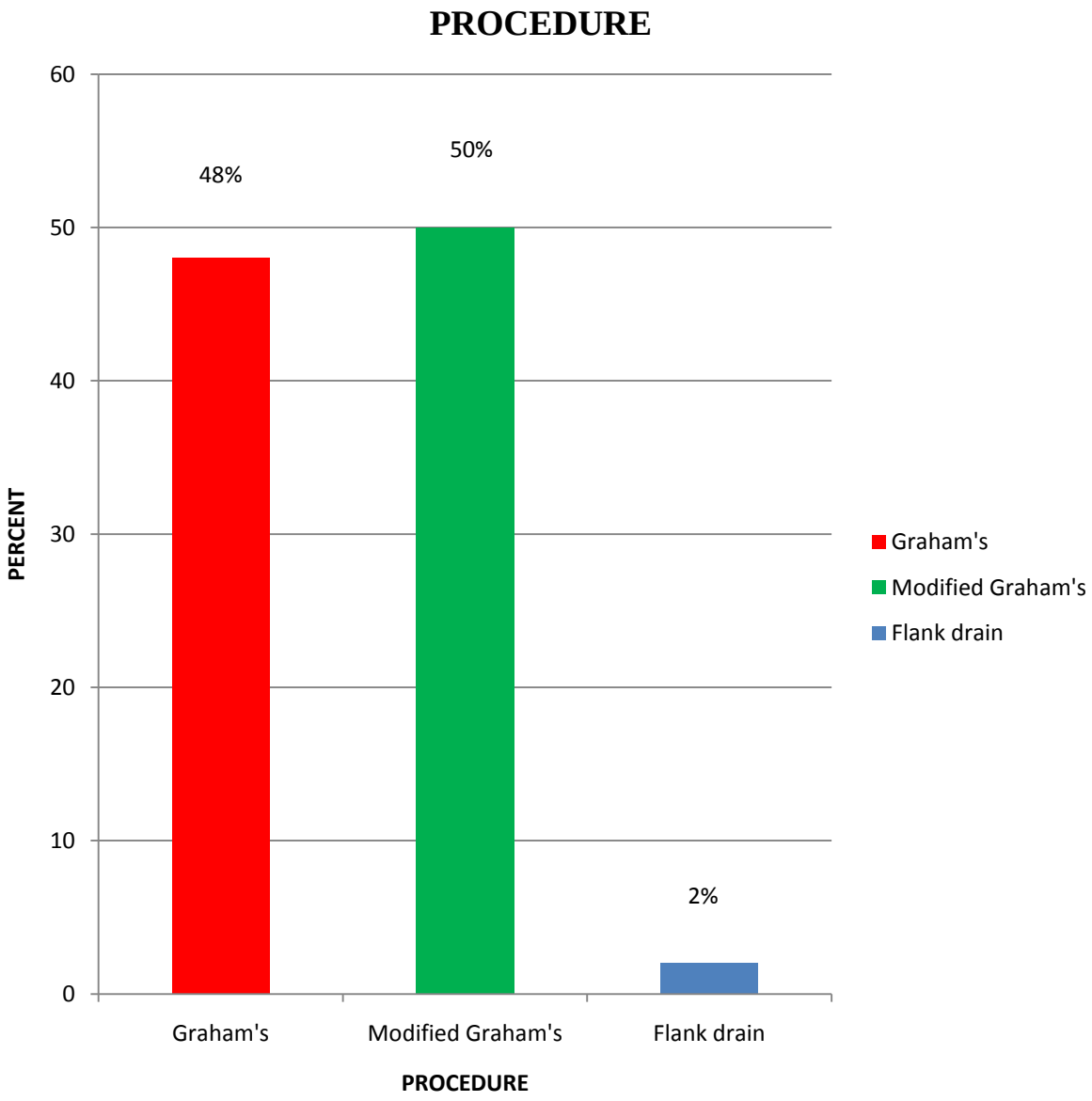
PROCEDURE

Procedure	Mortality		Morbidity					Total
			<= 7 days	8-14 days	15-21 days	22-28 days	>= 29 days	
Graham's	Death	Count	8	0	0	0	0	8
		% of Total	16.7%	.0%	.0%	.0%	.0%	16.7%
	Discharge	Count	4	28	5	2	1	40
		% of Total	8.3%	58.3%	10.4%	4.2%	2.1%	83.3%
Modified Graham's	Death	Count	12	2	0			14
		% of Total	24.0%	4.0%	.0%			28.0%
	Discharge	Count	3	31	2			36
		% of Total	6.0%	62.0%	4.0%			72.0%
Flank drain	Death	Count	2					2
		% of Total	100.0%					100.0%

The frequency of the Procedure, Mortality and Morbidity has been cross tabulated above. To assess if the Procedure has any significant effect on the Morbidity and Mortality has been exhibited below. The result of the Multivariate analysis shows that the Procedure has no significant effect on the Mortality and Morbidity of the patient.

Multivariate analysis

Source	Dependent Variable	Type III Sum of Squares	df	Mean Square	F	*Sig.*	Partial Eta Squared
Procedure	Mortality	1.493	2	.747	4.325	*.016*	.082
	Morbidity	3.130	2	1.565	3.188	*.046*	.062



SURGERY DURATION

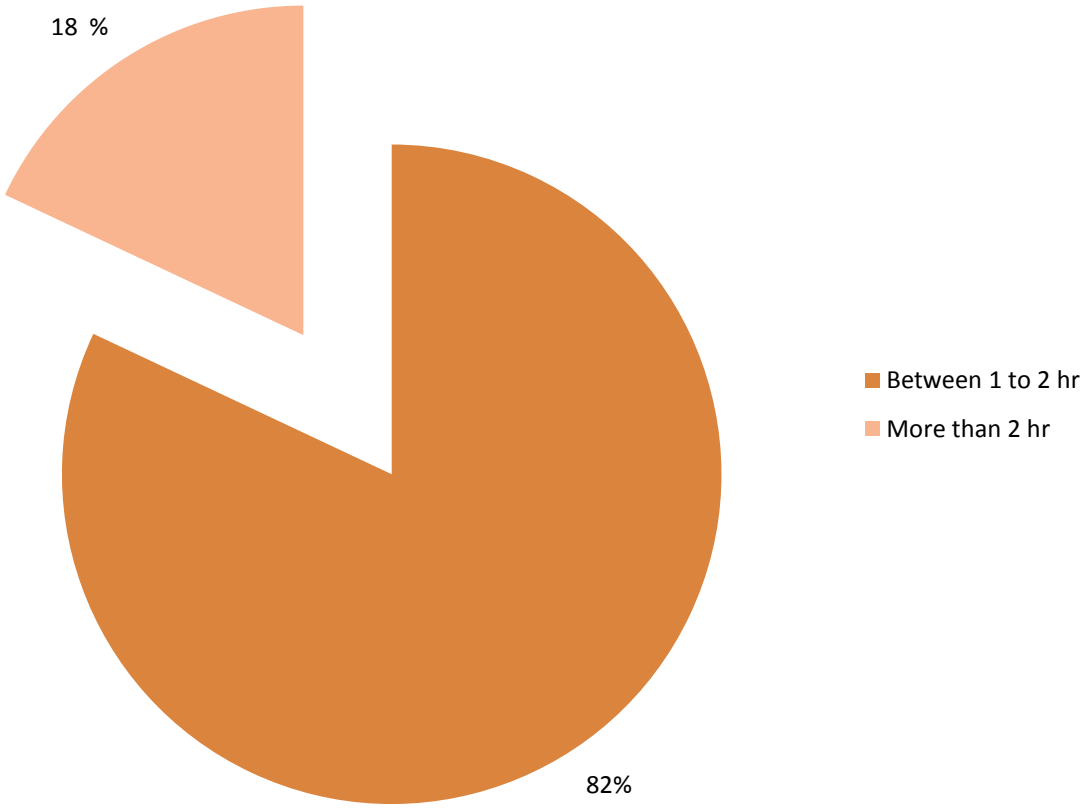
SurgeryDuration	Mortality		Morbidity					Total
			<= 7 days	8-14 days	15-21 days	22-28 days	>= 29 days	
Between 1 to 2 hr	Death	Count	14	2	0	0	0	16
		% of Total	17.1%	2.4%	.0%	.0%	.0%	19.5%
	Discharge	Count	7	50	6	2	1	66
		% of Total	8.5%	61.0%	7.3%	2.4%	1.2%	80.5%
More than 2 hr	Death	Count	8	0	0			8
		% of Total	44.4%	.0%	.0%			44.4%
	Discharge	Count	0	9	1			10
		% of Total	.0%	50.0%	5.6%			55.6%

The frequency of the Duration of the surgery, Mortality and Morbidity has been cross tabulated above. To assess if the Surgery duration has any significant effect on the Morbidity and Mortality has been exhibited below. The result of the Multivariate analysis shows that the Surgery duration has significant effect on the Mortality of the patient (sig < 0.05). The size of the effect on the Mortality is 5.0 %. The surgery duration has no significant effect on the Morbidity of the patient as the significant value is less than 5 %.

Multivariate analysis

Source	Dependent Variable	Type III Sum of Squares	df	Mean Square	F	*Sig.*	Partial Eta Squared
Duration surgery	Mortality	.918	1	.918	5.191	*.025*	.050
	Morbidity	1.253	1	1.253	2.480	*.119*	.025

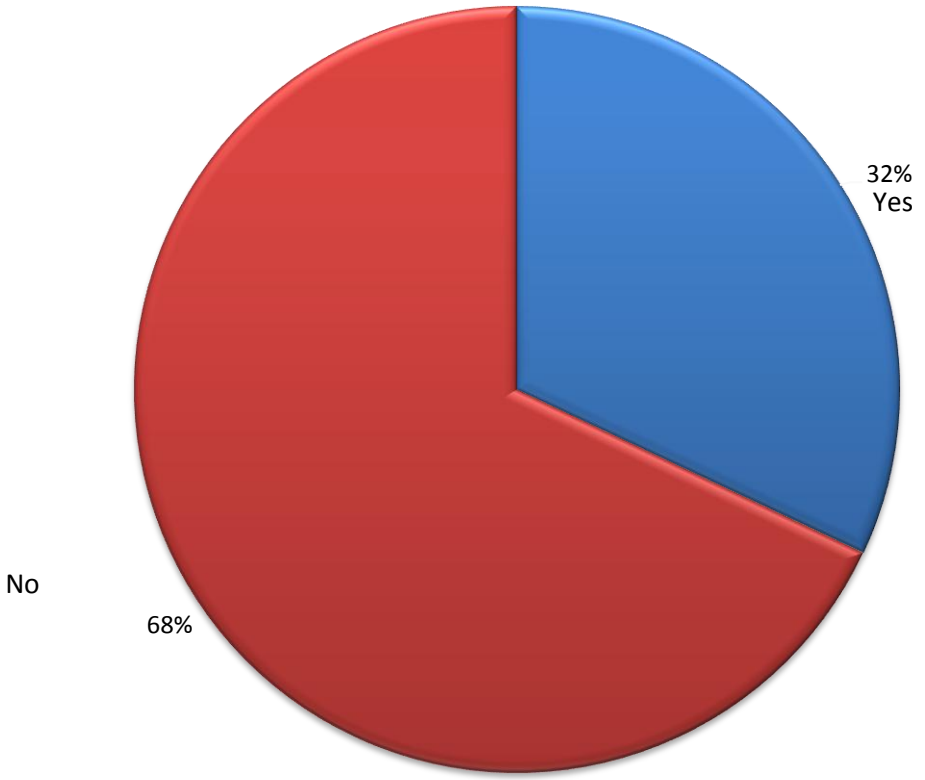
DURATION SURGERY



RESPIRATORY SUPPORT

Respiratory support	No. of Patients	Percent
Yes	32	32.0
No	68	68.0
Total	**100**	**100.0**

RESPIRATORY SUPPORT



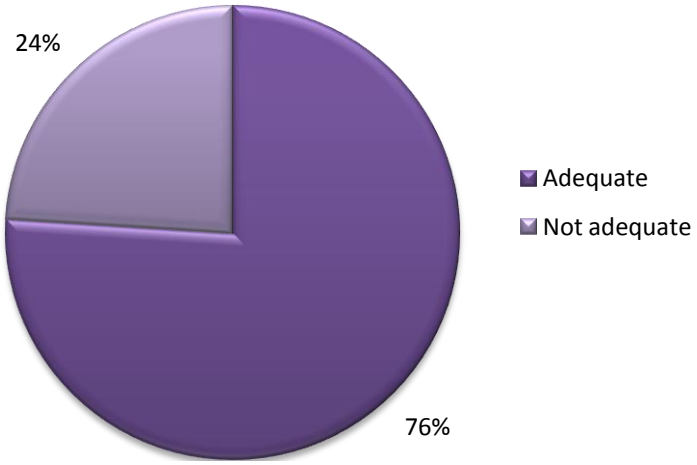
CIRCULATORY SUPPORT

Circulatory Support	No. of Patients	Percent
Yes	24	24.0
No	76	76.0
Total	**100**	**100.0**

RENAL FUNCTION

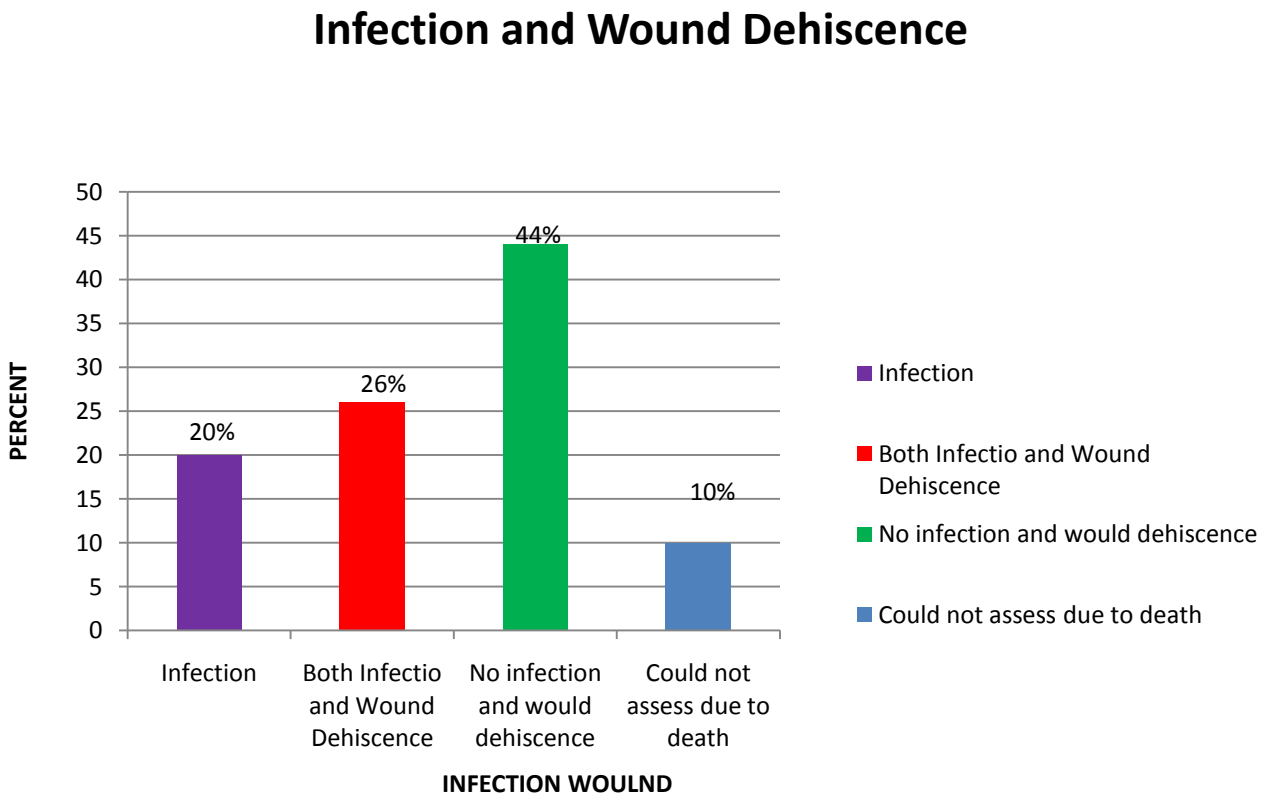
Renal function	No. of Patients	Percent
Adequate	76	76.0
Not adequate	24	24.0
Total	**100**	**100.0**

RENAL FUNCTION



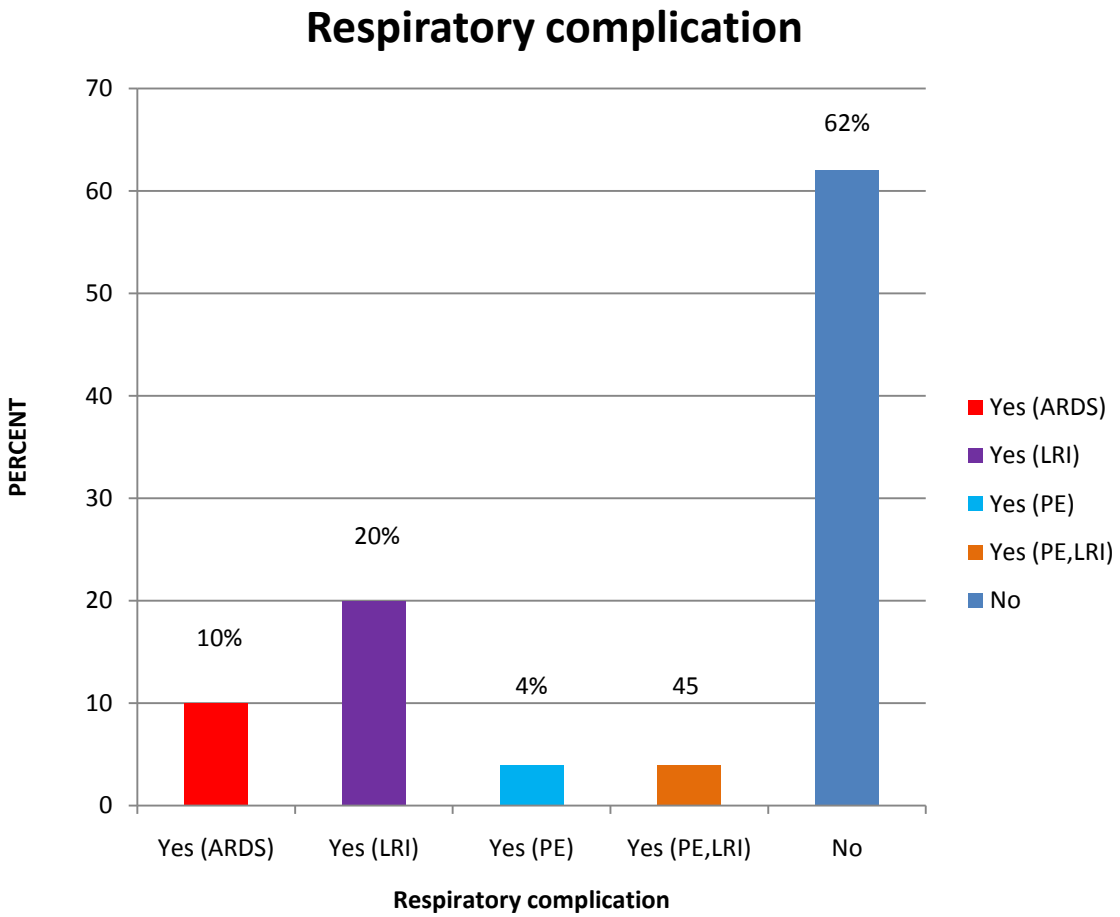
INFECTION AND WOUND DEHISCENCE

Infection and wound dehiscence	No. of Patients	Percent
Infection	20	20.0
Both Infection and Wound Dehiscence	26	26.0
No infection and would dehiscence	44	44.0
Could not assess due to death	10	10.0
Total	**100**	**100.0**



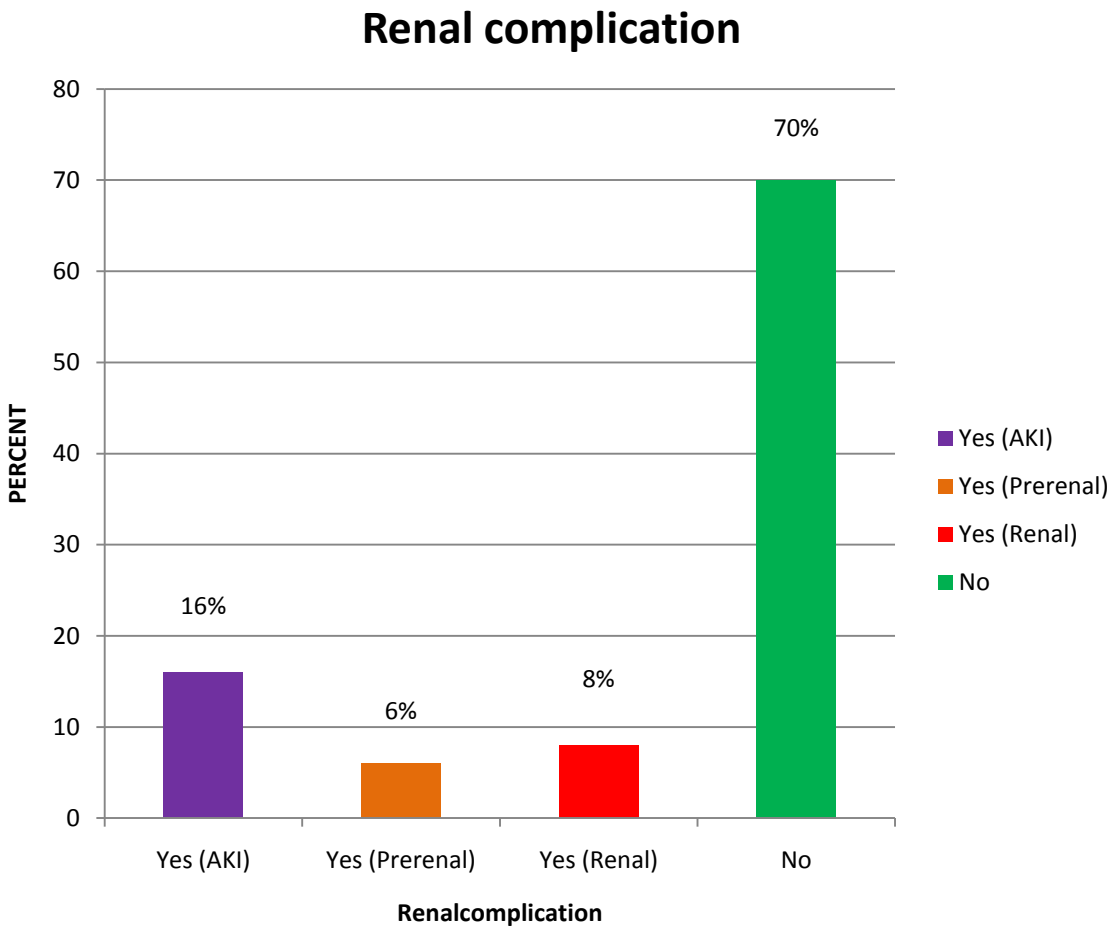
RESPIRATORY COMPLICATION

Respiratory complication		No. of Patients	Percent
YES	ARDS	10	10.0
	LRI	20	20.0
	PE	4	4.0
	PE,LRI	4	4.0
NO		62	62.0
Total		**100**	**100.0**



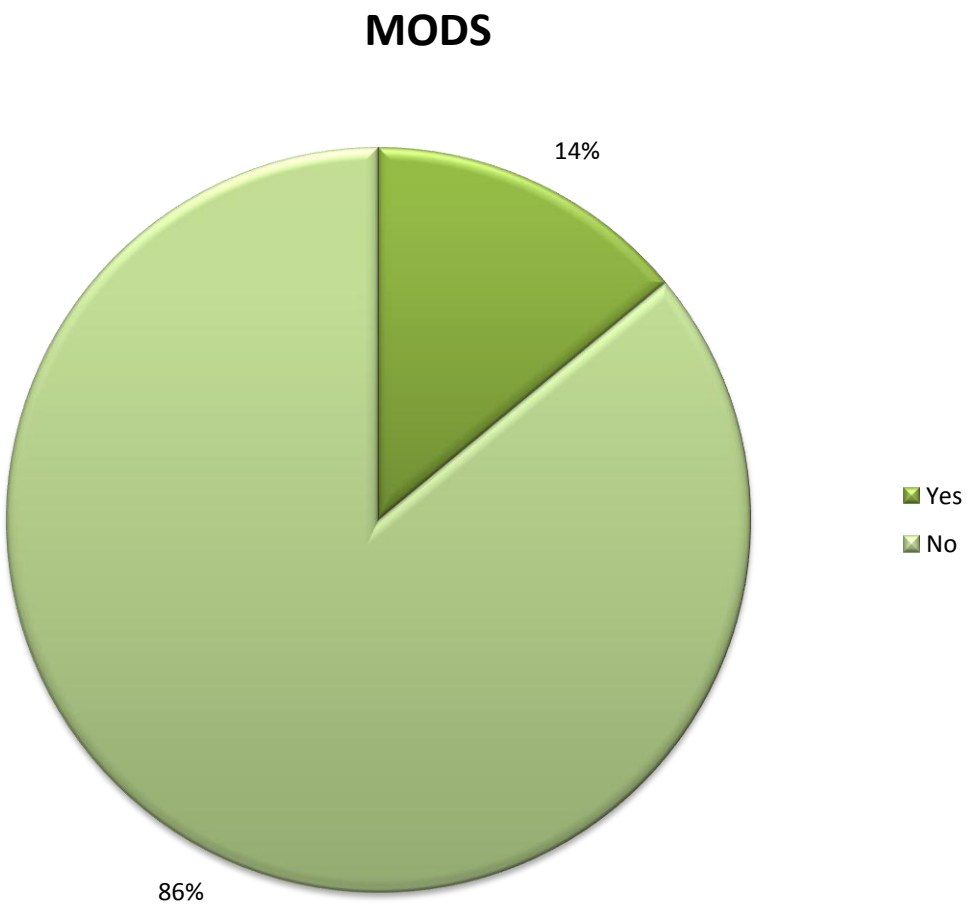
RENAL COMPLICATION

Renal complication		No. of Patients	Percent
YES	AKI	16	10.0
	Prerenal	6	20.0
	Renal	8	4.0
NO		62	62.0
Total		**100**	**100.0**



MODS

MODS	No. of Patients	Percent
YES	14	14.0
NO	86	86.0
Total	**100**	**100.0**



CAUSE OF DEATH

Cause of death	Frequency	Percent
AKI	4	16.7
ARDS	1	4.2
MODS	10	41.7
SEPTIC SHOCK	9	37.5
Total	**24**	**100.0**

CAUSE OF DEATH AND DURATION OF HOSPITAL STAY

CAUSE OF DEATH	DURATION OF HOSPITAL STAY					Total
	<= 7 days	8-14 days	15-21 days	22-28 days	>= 29 days	
AKI	2	2	0	0	0	4
ARDS	1	0	0	0	0	1
MODS	10	0	0	0	0	10
SEPTIC SHOCK	9	0	0	0	0	9
%	**91.7%**	**8.3%**	**-**	**-**	**-**	**24**

CROSS TABULATION ON MORBIDITY (DURATION OF HOSPITAL STAY) AND FINAL OUTCOME

CROSS TABULATION ON MORBIDITY AND FINAL OUTCOME								
			Morbidity					Total
Final Outcome	Final outcome		<=7 days	8-14 days	15-21 days	22-28 days	>= 29 days	
	Death	Count	22	2	0	0	0	**24**
		% of Total	22.0%	2.0%	.0%	.0%	.0%	**24.0%**
	Discharge	Count	7	59	7	2	1	**76**
		% of Total	7.0%	59.0%	7.0%	2.0%	1.0%	**76.0%**

DISCUSSION

- Age of 60 years and above had mortality of 46% and also this mortality is high within the first 48 hrs and up to 7 days. Average duration of hospital stay was 7 to 14 days which includes 59% regarding all age group. Multivariate analysis showed age has significant effect on the mortality (P = 0.000) and morbidity (P = 0.026).
- Female sex was more related to severe disease and mortality (100%).
- Most of the patients initially presented with epigastric pain (86%).
- Duration of pain of 3 or more days when presenting to hospital had related to mortality. Duration of 3 days had 35.7% and 4 or more 50% mortality. Duration of pain has significant effect on mortality (P = 0.15)
- 82% of the patients had past history of peptic ulcer and on and off treatment.
- Only 16% of patients had history of NSAID usage before perforation.
- 48% of patients had the habit of both smoking and alcohol and 40% are neither smoking nor alcoholic.
- On physical examination 96% had respiratory distress, 80% had anemia, 38% had tachycardia, 26% both hypotension and tachycardia, and 36% had normal pulse and blood pressure.

- Patients who had Class 3 shock at the time of admission had 66% mortality. Shock has significant effect on both morbidity (P = 0.001) and mortality (P = 0.000).
- Preoperative lower respiratory infection had 40% mortality. Preoperative LRI had significant effect on mortality (P = 0.042).
- Biochemical analysis of serum showed 62% high urea and 46% high creatinine. Hyponatremia and hypokalemia was seen in 20% and 14% of the patients.
- Hematological evaluation showed 72% of patients had normal leucocyte count but more towards higher end and 76% had high neutrophill percentage around 90%.
- On examination of abdomen 94% had signs of peritonitis and obliteration of liver dullness in 80% of patients.
- Chest Xray showed air under diaphragm in 76% of the patients.
- Duration between pain and surgery that is preoperative delay of more than 48 hours has significant effect on mortality (P = 0.042) and morbidity (P = 0.043). 48 to 72 hours had 31% and more than 72 hrs had 32% mortality.
- Duration between admission and surgery related to final outcome is influenced by duration between initial pain and surgery.

- ASA score of 4 and 5 had 100% mortality and score of 3 had 64%. It has significant on mortality (P = 0.000) and morbidity (P = 0.000)
- Gastric perforation (30%) has more mortality than duodenal (20%) perforation.
- Size of perforation more than 1 cm has 42% mortality and it has significant effect on mortality and morbidity
- Contamination of more than 1 litre had significantly influences in mortality (P = 0.004) and morbidity (P = 0.0070. In our observation more than 1 litre related to 44% of death.
- Duration of the surgery more than 2 hours had significant effect on the mortality (P = 0.025)
- 32% of the patients were not extubated and continued on ventilator support and 24% were in need of circulatory support.
- 76% of patients had adequate renal function.
- 46% of the patients developed wound infection and 26% had wound dehiscence and underwent secondary suturing. No wound infection in 44%.
- Post operatively 20% patients had lower respiratory infection and 10% had ARDS leading to death.
- 24% of the patient had acute kidney injury and treated with supportive treatment. 6% had prerenal failure and treated with intravenous fluids.

- 14% of patients had developed multiple organ dysfunctions and died.
- Sepsis leading to shock and multiple organ dysfunctions is a cause of death.

CONCLUSION

THE FOLLOWING FACTORS WERE ASSOCIATED WITH MORBIDITY AND MORTALITY

- Age more than 60 years
- Duration between initial pain and surgery of more than 48 hours
- Class III or more shock
- ASA score of 3and more
- Size of more than 1 cm
- Contamination of more than 1 litre

THE FOLLOWING FACTORS WERE ASSOCIATED WITH MORTALITY

- Preoperative lower respiratory infection
- Duration of the surgery
- Post operative lower respiratory infection
- Acute respiratory distress syndrome

BIBLIOGRAPHY

1. Chalya PL, Mabula JB, Koy M, McHembe MD, Jaka HM, Kabangila R, et al. Clinical profile and outcome of surgical treatment of perforated peptic ulcers: A tertiary hospital experience. World J Emerg Surg 2011;6:31.
2. Mäkelä J, Laitinen S, Kairaluoma MI. Complications of peptic ulcer disease before and after the introduction of H2-receptor antagonists. Hepatogastroenterology 1992;39:144-148.
3. Noguiera C, Silva AS, Santos JN, Silva AG, Ferreira J, Matos E, et al. Perforated peptic ulcer: main factors of morbidity and mortality. World J Surg 2003;27:782-787.
4. Scheeres DE, DeKryger LL, Dean RE. Surgical treatment of peptic ulcer disease before and after introduction of H2 blockers. Am Surg 1987;53:392-395.
5. Imhof M, Epstein S, Ohmann C, Röher HD. Duration of survival after peptic ulcer perforation. World J Surg 2008;32:408- 412
6. Rahman MM, Islam MS, Flora S, Akhter SF, Hossain S, Karim F. Mortality in perforated peptic ulcer patients after selective management of stratified poor risk cases. World J Surg 2007;31:2341-2344.Baron JH: Paintress, princess and physician's paramour: poison or perforation? J R Soc Med 1998, 91(4):213-216.
7. Baron JH: Peptic ulcer. The mount sinai journal of medicine 2000, 67(1):58-62.
8. Baron JH, Sonnenberg A: Publications on peptic ulcer in Britain, France, Germany and the US. Eur J Gastroenterol Hepatol 2002, 14(7):711-715.
9. Schein M: Perforated peptic ulcer. In: Schein's common sense emergency abdominal surgery. vol. part III: Springer Berlin Heidelberg; 2005: 143-150.
10. Rayner HH: Treatment of perforated peptic ulcer. The Lancet 1930(11):107-108.
11. Sangster AH: Perforated peptic ulcer. An analysis of 100 consecutive cases. The Lancet 1939, 23:1311-1313.
12. Berson HL: Acute perforated peptic ulcers. An eighteen-year survey. American journal of surgery 1942, 16(2):385-394.
13. Hastings N, Machida R: Perforated peptic ulcer: results after simple surgical closure. American journal of surgery 1961, 102:136-142.
14. Conservative management of perforated peptic ulcer. The Lancet 1989, 16:1429-1430.

15. Lau WY, Leow CK: History of perforated duodenal and gastric ulcers. World journal of surgery 1997, 21(8):890-896.
16. Birks PM: Perforated peptic ulcer reated without operation. The Lancet 1947(5):467-468.
17. Lagoo S, McMahon RL, Kakihara M, Pappas TN, Eubanks S: The sixth decision regarding perforated duodenal ulcer. Jsls 2002, 6(4):359-368.
18. Lau WY: Perforated peptic ulcer: open versus laparoscopic repair. Asian J Surg 2002, 25(4):267-269.
19. Fujii Y, Asato M, Taniguchi N, Shigeta K, Omoto K, Itoh K, Suzukawa M: Sonographic diagnosis and successful nonoperative management of sealed perforated duodenal ulcer. J Clin Ultrasound 2003, 31(1):55-58.
20. Ramakrishnan K, Salinas RC: Peptic ulcer disease. Am Fam Physician 2007, 76(7):1005-1012.
21. Lunevicius R, Morkevicius M: Management strategies, early results, benefits, and risk factors of laparoscopic repair of perforated peptic ulcer. World journal of surgery 2005, 29(10):1299-1310.
22. Sivri B: Trends in peptic ulcer pharmacotherapy. Fundam Clin Pharmacol 2004, 18(1):23-31.
23. Druart ML, Van Hee R, Etienne J, Cadiere GB, Gigot JF, Legrand M, Limbosch JM, Navez B, Tugilimana M, Van Vyve E et al: Laparoscopic repair of perforated duodenal ulcer. A prospective multicenter clinical trial. Surg Endosc 1997, 11(10):1017-1020.
24. Zittel TT, Jehle EC, Becker HD: Surgical management of peptic ulcer disease today-indication, technique and outcome. Langenbecks Arch Surg 2000, 385(2):84-96.
25. Imhof M, Epstein S, Ohmann C, Roher HD: Duration of survival after peptic ulcer perforation. World journal of surgery 2008, 32(3):408-412.
26. Sarosi GA, Jr., Jaiswal KR, Nwariaku FE, Asolati M, Fleming JB, Anthony T: Surgical therapy of peptic ulcers in the 21st century: more common than you think. American journal of surgery 2005, 190(5):775-779.
27. Harbison SP, Dempsey DT: Peptic ulcer disease. Current problems in surgery 2005,42(6):346-454.
28. Bucher P, Oulhaci W, Morel P, Ris F, Huber O: Results of conservative treatment for perforated gastroduodenal ulcers in patients not eligible for surgical repair. Swiss Med Wkly 2007, 137(23-24):337-340.

29. Ahmed N: 23 years of the discovery of Helicobacter pylori: is the debate over? Ann Clin Microbiol Antimicrob 2005, 4:17.
30. Fischbach LA, Goodman KJ, Feldman M, Aragaki C: Sources of variation of Helicobacter pylori treatment success in adults worldwide: a meta-analysis. Int J Epidemiol 2002, 31(1):128-139.
31. Donovan AJ, Berne TV, Donovan JA: Perforated duodenal ulcer: an alternative therapeutic plan. Arch Surg 1998, 133(11):1166-1171.
32. Malfertheiner P, Megraud F, O'Morain C, Bazzoli F, El-Omar E, Graham D, Hunt R, Rokkas T, Vakil N, Kuipers EJ: Current concepts in the management of Helicobacter pylori infection: the Maastricht III Consensus Report. Gut 2007, 56(6):772-781.
33. Crofts TJ, Park KG, Steele RJ, Chung SS, Li AK: A randomized trial of nonoperative treatment for perforated peptic ulcer. N Engl J Med 1989, 320(15):970-973.
34. Truscott B, Withycombe JFR: Perforated peptic ulcer. An assessment of the value of nonoperative treatment. The Lancet 1950, 13:894-896.
35. Schein M: To drain or not to drain? The role of drainage in the contaminated and infected abdomen: an international and personal perspective. World journal of surgery 2008,32(2):312-321.
36. Siu WT, Leong HT, Law BK, Chau CH, Li AC, Fung KH, Tai YP, Li MK: Laparoscopic repair for perforated peptic ulcer: a randomized controlled trial. Annals of surgery 2002, 235(3):313-319.
37. Cellan-Jones CJ: A rapid method of treatment in perforated duodenal ulcer. BMJ 1929(36):1076-1077.
38. Graham R, R: The treatment of perforated duodenal ulcers. . Surg gynecol Obstet1937(64):235-238.
39. Fallat ME, White MJ, Richardson JD, Flint LM: Reassessment of Graham-Steele closure in acute perforated peptic ulcer. South Med J 1983, 76(10):1222-1224.
40. Whiteside OJ, Tytherleigh MG, Thrush S, Farouk R, Galland RB: Intra-operative peritoneal lavage--who does it and why? Annals of the Royal College of Surgeons of England 2005, 87(4):255-258.
41. Pai D, Sharma A, Kanungo R, Jagdish S, Gupta A: Role of abdominal drains in perforated duodenal ulcer patients: a prospective controlled study. Aust N Z J Surg 1999, 69(3):210-213.
42. Branicki FJ: Abdominal emergencies: diagnostic and therapeutic laparoscopy. Surg Infect (Larchmt) 2002, 3(3):269-282.

43. Schwesinger WH, Page CP, Sirinek KR, Gaskill III HV, Melnick G, Strodel WE: Operations for peptic ulcer disease: paradigm lost. J Gastrointest Surg 2001, 5:438-443.
44. Chandra SS, Kumar SS: Definitive or conservative surgery for perforated gastric ulcer? –An unresolved problem. International Journal of Surgery 2009:1-4.
45. Jordan PH, Jr., Thornby J: Perforated pyloroduodenal ulcers. Long-term results with omental patch closure and parietal cell vagotomy. Annals of surgery 1995, 221(5):479-486; discussion 486-478.
46. Ates M, Coban S, Sevil S, Terzi A: The efficacy of laparoscopic surgery in patients with peritonitis. Surgical laparoscopy, endoscopy & percutaneous techniques 2008, 18(5):453-456.
47. Lau H: Laparoscopic repair of perforated peptic ulcer: a meta-analysis. Surg Endosc 2004,18(7):1013-1021.
48. Lunevicius R, Morkevicius M: Risk factors influencing the early outcome results after laparoscopic repair of perforated duodenal ulcer and their predictive value. Langenbecks Arch Surg 2005, 390(5):413-420.
49. Bertleff MJ, Liem RS, Bartels HL, Robinson PH, Van der Werff JF, Bonjer HJ, Lange JF: The "stamp method": a new treatment for perforated peptic ulcer? Surg Endosc 2006,20(5):791-793.
50. Alvarado-Aparicio HA, Moreno-Portillo M: Multimedia article: management of duodenal ulcer perforation with combined laparoscopic and endoscopic methods. Surg Endosc 2004, 18(9):1394.
51. Nelson R, Edwards S, Tse B: Prophylactic nasogastric decompression after abdominal surgery. Cochrane database of systematic reviews (Online) 2007(3):CD004929.
52. Daryaei P, Vaghef Davari F, Mir M, Harirchi I, Salmasian H: Omission of nasogastric tube application in postoperative care of esophagectomy. World journal of surgery 2009, 33(4):773-777.
53. St Peter SD, Valusek PA, Little DC, Snyder CL, Holcomb GW, 3rd, Ostlie DJ: Does routine nasogastric tube placement after an operation for perforated appendicitis make a difference? The Journal of surgical research 2007, 143(1):66-69.
54. Siu WT, Chau CH, Law BK, Tang CN, Ha PY, Li MK: Routine use of laparoscopic repair for perforated peptic ulcer. The British journal of surgery 2004, 91(4):481-484.

55. Waddell TK, Rotstein OD: Antimicrobial prophylaxis in surgery. Committee on Antimicrobial Agents, Canadian Infectious Disease Society. Cmaj 1994, 151(7):925-931.
56. Lunevicius R, Morkevicius M: Comparison of laparoscopic versus open repair for perforated duodenal ulcers. Surg Endosc 2005, 19(12):1565-1571.
57. Lam PW, Lam MC, Hui EK, Sun YW, Mok FP: Laparoscopic repair of perforated duodenal ulcers: the"three-stitch" Graham patch technique. Surg Endosc 2005, 19(12):1627-1630.
58. Gupta S, Kaushik R, Sharma R, Attri A: The management of large perforations of duodenal ulcers. BMC Surg 2005, 5:15.
59. Rahuman MM, Saha AK, Rahim A: Experience of peptic ulcer perforation over a decade in a teaching hospital of southern Bangladesh. Ceylon Med J 2003, 48(2):53-55.
60. Sharma SS, Mamtani MR, Sharma MS, Kulkarni H: A prospective cohort study of postoperative complications in the management of perforated peptic ulcer. BMC Surg 2006, 6:8.
61. Feliciano DV, Bitondo CG, Burch JM, Mattox KL, Jordan GL, Jr., DeBakey ME: Emergency management of perforated peptic ulcers in the elderly patient. American journal of surgery 1984, 148(6):764-767.
62. Blomgren LG: Perforated peptic ulcer: long-term results after simple closure in the elderly. World journal of surgery 1997, 21(4):412-414; discussion 414-415. Svanes C. Trends in perforated peptic ulcer: incidence, etiology, treatment, and prognosis. World J Surg. 2000 Mar;24(3):277-83.
63. Joyce M. Piper, DrPH; Wayne A. Ray, PhD; James R. Daugherty, MS; and Marie R. Griffin, MD, MPH Corticosteroid Use and Peptic Ulcer Disease: Role of Nonsteroidal Anti-inflammatory Drugs *Ann Intern Med*. 1991;114(9):735-740. doi:10.7326/0003-4819-114-9-735

PROFORMA

Name : _____ IP. No: Age :

Address: _____ Sex :

_____

Occupation:	
DOA & Time:	
DOS & Time:	
DOD:	
Chief complaints: Abdominal pain site	
Started on & Time:	
Treatment history: For present illness: Yes/No	
Type of treatment:	
Duration:	
Past history: Peptic ulcer disease:	
Drugs used:	
Surgery for peptic ulcer:	
Personal history: Smoking, duration:	
Alcohol, duration:	
Dietary habit:	
Co morbid illness: HT / DM / CLD / CRF / TB	
COPD/CVA	
General examination	
Conscious	
Orientation	

Hydration		
Fever		
Jaundice		
Anemia		
Respiratory distress		
Vitals		
PR:		
BP:		
Temp:		
RR:		
Systemic examination:		
CVS:		
RS:		
Abdomen		
Investigations :		
CXR		
USG		
Biochemistry	Glucose	
	Urea	
	creatinine	
	Na+	
	K+	
CBC	TC:	
	DC:	
	Platelets:	

DIAGNOSIS	
Treatment:	
PRE operative: Peritonitis	
Shock	
ASA score	
Duration between pain and surgery:	
PER operative:	
Surgery : open / lap	
Site of perforation	
Size : <1cm / 1 – 3cm / >3cm	
Contamination	
Procedure done	
Duration of procedure	
Drainage	
POST Operative period	
Respiratory support	
Circulatory support	
Renal function	
Complications :	
Leakage	
Fluid collection	
Paralytic ileus	
Intestinal obstruction	
Bleeding	
Wound complication	

Pulmonary complication:	
Cardiac complication	
Renal complication	
Hepatic complication	
Multi organ failure	
Others	
FINAL OUTCOME	

S.No	NAME	AGE	SEX	EPIGASTRIC PAIN	OTHER SITES	PAIN DURATION in DAYS	PAST history of PEPTIC ULCER	history of NSAID	SMOKING	ALCOHOL	COMORBID ILLNESS	RESPIRATORY DISTRESS	ANEMIA	FEVER	JAUNDICE	TACHYCARDIA	HYPOTENSION	SHOCK CLASS	Pre operaative LRI	CXR AIR under diaphargm	USG	CECT
1	Dhamodaran	16	M	Y		1	Y	Y	N	N	N	Y	N	Y	N	Y	Y	3	Y	Y		
2	Arasu	55	M	Y		1	Y	Y	Y	Y	PVD,BA	N	N	N	N	Y	N	2	Y	N		
3	Venkatesh	28	M	N	LIF	2	N	Y	Y	Y	HIV,HepB	Y	Y	Y	N	Y	Y	3	Y	Y		
4	Joseph	70	M	Y		3	Y	N	Y	Y	N	Y	Y	N	N	Y	Y	3	N	N		
5	Subramani	60	M	N	allover	2	Y	N	Y	N	N	Y	N	N	N	Y	N	2	N	Y		
6	Hussain	60	M	Y		10	Y	N	N	N	N	Y	Y	N	N	N	N	1	N	Y		
7	Narayanasamy	65	M	Y		2	N	N	Y	Y	N	N	N	N	N	N	N	1	N	Y		
8	Maruthu	40	M	Y		3	Y	N	N	N	N	N	N	N	N	N	N	1	N	Y		
9	Paneer selvam	56	M	Y		3	Y	N	N	N	N	N	N	N	N	Y	N	2	N	Y		
10	Kanimozhi	45	F	Y		3	N	Y	N	N	N	Y	Y	N	N	Y	Y	3	Y	N		
11	Palani	60	M	Y		7	Y	N	Y	Y	DCLD	Y	Y	N	Y	Y	Y	3	Y	Y		
12	Krishnamoorthy	55	M	Y		2	Y	N	Y	Y	N	Y	N	N	N	Y	Y	3	N	Y		
13	Selvam	60	M	Y		7	Y	N	Y	Y	N	Y	N	N	N	Y	N	2	Y	N		
14	Murugesan	30	M	N	Diffuse	1	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y		
15	Muna	25	M	Y		2	Y	N	Y	Y	N	N	N	N	N	N	N	N	N	Y		
16	Sriram	45	M	Y		2	Y	N	N	N	N	N	Y	N	N	Y	Y	3	N	Y		
17	Nagendran	50	M	Y		3	Y	N	Y	Y	N	N	N	N	N	Y	N	2	N	Y		
18	Kamaraj	38	M	Y		1	Y	N	Y	Y	BA	N	N	N	N	Y	Y	3	Y	Y		
19	Sighumalick	21	M	Y		3	Y	N	N	N	N	N	Y	Y	N	Y	N	2	N	Y		
20	Mani	55	M	Y		3	Y	N	Y	Y	N	N	N	N	N	Y	Y	2	N	Y		
21	Rangachari	65	M	Y		1	Y	N	Y	Y	DM, CRF	N	N	N	N	N	N	1	N	Y		
22	Pintu	25	M	Y		3	Y	N	N	N	N	N	N	N	N	N	N	1	N	Y		
23	Kumar	26	M	Y		3	Y	N	N	Y	N	N	N	N	N	Y	N	1	N	Y		
24	Siva	40	M	Y		1	Y	N	Y	Y	N	N	N	N	N	N	N	1	N	Y		
25	Arulraj	40	M	Y		3	Y	N	N	N	N	N	N	N	N	N	N	1	N	N		

S.No	NAME	AGE	SEX	EPIGASTRIC PAIN	OTHER SITES	PAIN DURATION in DAYS	PAST history of PEPTIC ULCER	history of NSAID	SMOKING	ALCOHOL	COMORBID ILLNESS	RESPIRATORY DISTRESS	ANEMIA	FEVER	JAUNDICE	TACHYCARDIA	HYPOTENSION	SHOCK CLASS	Pre operaative LRI	CXR AIR under diaphargm	USG	CECT
26	Kumar	40	M	Y		1	Y	N	Y	N	N	N	N	N	N	N	N	1	N	Y		
27	Paramasivam	41	M	N	RtHC	2	Y	N	N	N	N	N	N	Y	N	Y	N	1	N	N		
28	Masilamani	16	M	Y		1	N	Y	N	N	N	N	N	N	N	N	N	1	N	N		FA
29	Prakash	24	M	Y		3	Y	N	Y	Y	N	Y	N	N	N	Y	N	2	N	Y		
30	Saravanan	35	M	N	allover	2	Y	N	Y	Y	N	Y	N	N	N	Y	N	2	N	Y		
31	Sivakumar	25	M	Y		2	Y	N	N	N	N	N	N	N	N	N	N	1	N	Y		
32	Abshiek kumar	24	M	N	allover	2	Y	N	N	N	N	N	N	N	N	N	N	1	N	N	FF	
33	Gangan	35	M	Y		3	Y	N	N	N	N	N	N	N	N	N	N	1	N	Y		
34	Elumalai	40	M	Y		2	Y	N	Y	Y	N	N	N	N	N	Y	N	1	N	Y		
35	Manikandan	22	M	Y		1	Y	N	N	N	N	N	N	N	N	N	N	1	N	Y		
36	Kumar	37	M	Y		6hrs	y	n	Y	Y	N	N	N	N	N	Y	N	2	N	Y		
37	Ettiyappan	70	M	N	allover	4	Y	N	N	Y	N	N	N	N	N	Y	N	2	N	N	FF	
38	Devaraj	54	M	Y		4	Y	N	Y	Y	N	Y	N	N	N	Y	Y	3	N	Y		
39	Babu	40	M	Y		1	Y	N	Y	Y	N	N	N	N	N	Y	N	2	N	Y		
40	Murugan	53	M	Y		1	N	N	N	N	N	N	N	N	N	N	N	2	N	Y		
41	Santhanam	23	M	Y		3	Y	N	Y	Y	N	N	N	N	N	Y	N	1	N	Y		
42	Arumugam	48	M	Y		1	N	N	N	N	N	N	N	N	N	Y	N	1	N	N		
43	Fakrudeen	79	M	Y		1	Y	N	N	N	N	Y	N	N	N	Y	Y	3	N	Y		
44	Anbarasan	25	M	Y		1	Y	Y	N	N	N	Y	N	Y	N	Y	Y	3	Y	Y		
45	Jeyachandran	55	M	Y		1	Y	Y	Y	Y	BA	N	N	N	N	Y	N	2	Y	N		
46	Rajagopal	28	M	Y		2	N	Y	Y	Y	N	Y	Y	Y	N	Y	Y	3	Y	Y		
47	Siva	70	M	Y		3	Y	N	Y	Y	N	Y	Y	N	N	Y	Y	3	N	N		
48	Kasi	60	M	Y		2	Y	N	Y	N	N	Y	N	N	N	Y	N	2	N	Y		
49	Baskar	60	M	Y		10	Y	N	N	N	N	Y	Y	N	N	N	N	1	N	Y		
50	Bala	65	M	Y		2	N	N	Y	Y	N	N	N	N	N	N	N	N	N	Y		

S.No	NAME	AGE	SEX	EPIGASTRIC PAIN	OTHER SITES	PAIN DURATION in DAYS	PAST history of PEPTIC ULCER	history of NSAID	SMOKING	ALCOHOL	COMORBID ILLNESS	RESPIRATORY DISTRESS	ANEMIA	FEVER	JAUNDICE	TACHYCARDIA	HYPOTENSION	SHOCK CLASS	Pre operaative LRI	CXR AIR under diaphargm	USG	CECT
51	Ramadoss	18	M	Y		1	Y	Y	N	N	N	Y	N	Y	N	Y	Y	3	Y	Y		
52	Lakshmanan	56	M	Y		1	Y	Y	Y	Y	PVD,BA	N	N	N	N	Y	N	2	Y	N		
53	Vishalkumar	28	M	N	LIF	2	N	Y	Y	Y	HIV,HepB	Y	Y	Y	N	Y	Y	3	Y	Y		
54	Krishnapillai	72	M	Y		3	Y	N	Y	Y	N	Y	Y	N	N	Y	Y	3	N	N		
55	Raj	60	M	N	Allover	2	Y	N	Y	N	N	Y	N	N	N	Y	N	2	N	Y		
56	Radhakrishnan	60	M	Y		10	Y	N	N	N	N	Y	Y	N	N	N	N	1	N	Y		
57	Appuraj	67	M	Y		2	N	N	Y	Y	N	N	N	N	N	N	N	N	N	Y		
58	Purusothaman	40	M	Y		3	Y	N	N	N	N	N	N	N	N	N	N	N	N	Y		
59	Gopal	58	M	Y		3	Y	N	N	N	N	N	N	N	N	Y	N	2	N	Y		
60	Raji	48	F	Y		3	N	Y	N	N	N	Y	Y	N	N	Y	Y	3	Y	N		
61	Ravikumar	60	M	Y		7	Y	N	Y	Y	DCLD	Y	Y	N	Y	Y	Y	3	Y	Y		
62	Gnanamoorthy	56	M	Y		2	Y	N	Y	Y	N	Y	N	N	N	Y	Y	3	N	Y		
63	Moorthy	62	M	Y		7	Y	N	Y	Y	N	Y	N	N	N	Y	N	2	Y	N		
64	Yesuraj	34	M	N	Diffuse	1	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y		
65	Jeyaraman	28	M	Y		2	Y	N	Y	Y	N	N	N	N	N	N	N	N	N	Y		
66	Balaji	49	M	Y		2	Y	N	N	N	N	N	Y	N	N	Y	Y	3	N	Y		
67	Lingaiyan	54	M	Y		3	Y	N	Y	Y	N	N	N	N	N	Y	N	2	N	Y		
68	Srinivasan	42	M	Y		1	Y	N	Y	Y	BA	N	N	N	N	Y	Y	3	Y	Y		
69	Bindy	26	M	Y		3	Y	N	N	N	N	N	Y	Y	N	Y	N	2	N	Y		
70	Shankar	58	M	Y		3	Y	N	Y	Y	N	N	N	N	N	Y	Y	2	N	Y		
71	Sabiq	66	M	Y		1	Y	N	Y	Y	DM, CRF	N	N	N	N	N	N	1	N	Y		
72	Rangan	28	M	Y		3	Y	N	N	N	N	N	N	N	N	N	N	1	N	Y		
73	Kesavan	29	M	Y		3	Y	N	N	Y	N	N	N	N	N	Y	N	1	N	Y		
74	Premkumar	43	M	Y		1	Y	N	Y	Y	N	N	N	N	N	N	N	1	N	Y		
75	Ahamed	40	M	Y		3	Y	N	N	N	N	N	N	N	N	N	N	1	N	N		

S.No	NAME	AGE	SEX	EPIGASTRIC PAIN	OTHER SITES	PAIN DURATION in DAYS	PAST history of PEPTIC ULCER	history of NSAID	SMOKING	ALCOHOL	COMORBID ILLNESS	RESPIRATORY DISTRESS	ANEMIA	FEVER	JAUNDICE	TACHYCARDIA	HYPOTENSION	SHOCK CLASS	Pre operaative LRI	CXR AIR under diaphargm	USG	CECT
76	Manoharan	40	M	Y		1	Y	N	Y	N	N	N	N	N	N	N	N	1	N	Y		
77	Chandran	44	M	N	RtHC	2	Y	N	N	N	N	N	N	Y	N	Y	N	1	N	N		
78	Tarunkumar	18	M	Y		1	N	Y	N	N	N	N	N	N	N	N	N	1	N	N		FA
79	Rajendran	29	M	Y		3	Y	N	Y	Y	N	Y	N	N	N	Y	N	2	N	Y		
80	Suriyakumar	37	M	N	allover	2	Y	N	Y	Y	N	Y	N	N	N	Y	N	2	N	Y		
81	Sivaraj	26	M	Y		2	Y	N	N	N	N	N	N	N	N	N	N	1	N	Y		
82	Gokul	28	M	N	allover	2	Y	N	N	N	N	N	N	N	N	N	N	1	N	N	FF	
83	Pandian	39	M	Y		3	Y	N	N	N	N	N	N	N	N	N	N	1	N	Y		
84	Manimaran	40	M	Y		2	Y	N	Y	Y	N	N	N	N	N	Y	N	1	N	Y		
85	Sandhanagopal	22	M	Y		1	Y	N	N	N	N	N	N	N	N	N	N	1	N	Y		
86	Ganesan	37	M	Y		6HRS	y	n	Y	Y	N	N	N	N	N	Y	N	2	N	Y		
87	Ganapathy	70	M	N	allover	4	Y	N	N	Y	N	N	N	N	N	Y	N	2	N	N	FF	
88	Muruganantham	54	M	Y		4	Y	N	Y	Y	N	Y	N	N	N	Y	Y	3	N	Y		
89	Barani	34	M	Y		1	Y	N	Y	Y	N	N	N	N	N	Y	N	2	N	Y		
90	Nithyanandham	53	M	Y		1	N	N	N	N	N	N	N	N	N	N	N	2	N	Y		
91	Rajesh	23	M	Y		3	Y	N	Y	Y	N	N	N	N	N	Y	N	1	N	Y		
92	Ramkumar	48	M	Y		1	N	N	N	N	N	N	N	N	N	Y	N	1	N	N		
93	Abdulla	79	M	Y		1	Y	N	N	N	N	Y	N	N	N	Y	Y	3	N	Y		
94	Anduraj	25	M	Y		1	Y	Y	N	N	N	Y	N	Y	N	Y	Y	3	Y	Y		
95	Prabakaran	55	M	Y		1	Y	Y	Y	Y	BA	N	N	N	N	Y	N	2	Y	N		
96	Rajan	28	M	Y		2	N	Y	Y	Y	N	Y	Y	Y	N	Y	Y	3	Y	Y		
97	Smith	70	M	Y		3	Y	N	Y	Y	N	Y	Y	N	N	Y	Y	3	N	N		
98	Devanathan	60	M	Y		2	Y	N	Y	N	N	Y	N	N	N	Y	N	2	N	Y		
99	Mani	60	M	Y		10	Y	N	N	N	N	Y	Y	N	N	N	N	1	N	Y		
100	Periyasamy	65	M	Y		2	N	N	Y	Y	N	N	N	N	N	N	N	N	N	Y		

USG - Ultrasonagram
CT - Computerised Tomography
FF - Free fluid
FA - Free air

S.No	NAME	GLUCOSE	UREA	CREATININE	SODIUM	POTTASSIUM	TOTAL COUNT	NEUTROPHIL %	LYMPHOCYT %	HEMOGLOBIN %	PERITONITIS	ASA SCORE	DURATION between PAIN & SURGERY in HOURS	DURATION between ADMISSION & SURGERY in HOURS	SURGERY	perforation SITE	SIZE in CM	CONTAMINATION in LITRES	CONTENTS	PROCEDURE	DURATION OF SURGERY in hours
1	Dhamodaran	107	68	1.7	141	4.2	15700	74	15	11	Y	2	33	3.3	O	PP	0.5	0.5	B	MG	2
2	Arasu	96	22	0.8	137	4.6	9200	90	9	13	Y	2	32	7.3	O	A	0.5	1	B	MG	1.3
3	Venkatesh	96	69	2	145	5.1	2500	56	15	8	Y	3	52	2.45	O	A	0.5	1	B	MG	2.3
4	Joseph	86	65	2.1	146	4.8	7800	90	9	9	Y	3	57	2.3	O	D1	3	2	GAN	MG	1.3
5	Subramani	126	125	1.5	137	3.6	6900	91	6	12	Y	3	28	4	O	P	0.5	0.5	PUS	G	2.3
6	Hussain	110	28	1.1	140	4	9800	78	25	9	Y	2	10 DAYS	12	O	D1	0.5	0	B	G	2.3
7	Narayanasamy	126	36	1	142	3.9	10400	72	14	11	Y	2	58	8	O	D1	0.5	0.05	B	MG	2
8	Maruthu	110	26	0.9	131	3.6	7700	70	20	12	Y	2	75	2	O	D1	0.5	1.5	B	MG	2
9	Paneer selvam	68	132	4.2	138	5.4	14400	74	24	11	Y	3	82	10	O	A	1.5	0.8	PUS	G	2.5
10	Kanimozhi	106	106	3.2	130	5.9	15600	90	9	8	Y	5	96	13	O	A	1	2	B	G	2
11	Palani	90	56	2.1	140	3.7	12000	88	13	8	Y	3	7 days	3	O	A	0.5	3	B	MG	3
12	Krishnamoorthy	70	101	5.1	139	3	6400	90	7	11	Y	3	72	11	O	A	1	3	B	G	2
13	Selvam	91	74	1.6	136	3.5	7200	60	35	13	Y	2	7 days	7	O	PP	0.5	1	B	MG	1.3
14	Murugesan	98	28	0.8	138	3.7	11600	60	38	9.8	Y	2	26	1.3	O	D1	0.5	0.5	B	MG	2
15	Muna	114	44	1.1	139	3.3	9500	83	12	13	Y	2	52	5	O	D1	0.5	2.5	B	G	2
16	Sriram	60	34	1.1	140	3.4	7500	84	9	11	Y	2	53	5	O	D1	1	2	B	G	2
17	Nagendran	87	40	1	131	3.8	3700	90	8	13	Y	2	76	4.3	O	D1	0.3	0.8	B	G	2.3
18	Kamaraj	153	42	0.7	132	4.8	3400	60	34	13	Y	2	30	7	O	D1	0.5	2	B	G	1.5
19	Sighumalick	114	40	0.9	128	4.6	5600	90	7	12	Y	2	17	5	O	D1	1	0.5	B	G	1.3
20	Mani	90	45	2.1	136	4.6	11600	78	20	11	Y	2	39	3	O	A	0.5	1	B	G	1.3
21	Rangachari	69	98	3.2	142	5.8	6800	90	8	9.6	Y	2	30	18	O	PP	0.5	0.75	B	MG	1.3
22	Pintu	90	38	1	137	3.8	10400	68	30	11	Y	2	45	9	O	D1	0.5	1	B	G	1.3
23	Kumar	108	28	0.9	138	3.8	7100	77	15	10	Y	2	41	5	O	D1	1	1	B	G	2
24	Siva	108	26	0.9	135	3.6	10200	82	12	12	Y	2	30	5	O	D1	0.5	1	B	G	1
25	Arulraj	110	28	1.2	134	3	11000	63	30	12	Y	2	42	6	O	P	1	1	B	MG	2

S.No	NAME	GLUCOSE	UREA	CREATININE	SODIUM	POTTASSIUM	TOTAL COUNT	NEUTROPHIL %	LYMPHOCYT %	HEMOGLOBIN %	PERITONITIS	ASA SCORE	DURATION between PAIN & SURGERY in HOURS	DURATION between ADMISSION & SURGERY in HOURS	SURGERY	perforation SITE	SIZE in CM	CONTAMINATION in LITRES	CONTENTS	PROCEDURE	DURATION OF SURGERY in hours
26	Kumar	106	46	1.1	137	3.6	10600	90	9	11	Y	2	27	3	O	D1	0.5	1	B	G	1.3
27	Paramasivam	120	28	1.5	133	4.6	8100	80	12	12	N	2	51	3.15	O	D1	0.3	0.1	B	G	1.3
28	Masilamani	94	25	0.8	138	3.8	9700	76	20	12	N	1	31	7.45	O	A	0.4	0.3	B	MG	1.45
29	Prakash	132	28	0.8	138	4.8	11400	93	7	16	Y	1	76	3	O	D1	0.4	0.5	B	G	1.45
30	Saravanan	82	64	2.5	136	4	4000	96	5	14	Y	1	53	5	O	A	0.5	3	B	G	1.3
31	Sivakumar	98	28	0.9	133	3.7	10100	86	10	14	Y	1	52	4	O	A	0.5	0.5	B	MG	1.45
32	Abshiekkumar	80	44	1.3	142	3.8	10000	72	23	12	N	1	56	8	O	D1	0.5	0.3	B	MG	1.3
33	Gangan	132	30	1,3	139	3.2	5000	57	37	14	Y	1	75	4	O	D1	0.5	0.5	B	G	1.3
34	Elumalai	185	23	0.7	136	2.5	5900	63	33	13	Y	1	52	2	O	D1	1	1.5	B	MG	2
35	Manikandan	106	28	0.7	136	3.6	10500	72	27	11	Y	1	28	3.3	O	A	1	2	B	G	2
36	Kumar	96	36	1.5	142	4.1	5800	84	14	9.2	Y	1	12	5.3	O	D1	0.5	0.2	B	G	2
37	Ettiyappan	80	69	4.3	143	4	5500	60	35	6.3	Y	3	72	5.3	O	D1	0.5	2	B	MG	1.3
38	Devaraj	90	67	3	145	4	7800	89	11	7	Y	4	24	6	O	-	-	2	B	DRAIN	1
39	Babu	83	28	0.8	140	3.8	7200	88	10	11	Y	2	28	4	O	P	0.5	0.2	B	MG	1.2
40	Murugan	114	28	0.9	134	3.5	2600	70	29	16	Y	2	26	2	O	D1	0.7	1.5	B	MG	2
41	Santhanam	120	28	0.8	141	3	12100	80	10	9.6	Y	3	80	8	O	PP	0.3	1	B	MG	1.3
42	Arumugam	59	34	1.2	143	3.6	19700	92	6	13	Y	1	29	5.3	O	P	0.4	1	B	MG	2
43	Fakrudeen	67	155	2.9	136	5.2	15900	92	4	9.6	Y	2	30	5	O	D1	2	3	B	G	2
44	Anbarasan	107	68	1.7	141	4.2	15700	74	15	11	Y	2	33	3.3	O	PP	0.5	0.5	B	MG	2
45	Jeyachandran	96	22	0.8	137	4.6	9200	90	9	13	Y	2	32	7.3	O	A	0.5	1	B	MG	1.3
46	Rajagopal	96	69	2	145	5.1	2500	56	15	8	Y	3	52	2.45	O	A	0.5	1	B	MG	2.3
47	Siva	86	65	2.1	146	4.8	7800	90	9	9	Y	3	57	2.3	O	D1	3	2	GAN	MG	1.3
48	Kasi	126	125	1.5	137	3.6	6900	91	6	12	Y	3	28	4	O	P	0.5	0.5	PUS	G	2.3
49	Baskar	110	28	1.1	140	4	9800	78	25	9	Y	2	10 DAYS	12	O	D1	0.5	0	B	G	2.3
50	Bala	126	36	1	142	3.9	10400	72	14	11	Y	2	58	8	O	D1	0.5	0.05	B	MG	2

S.No	NAME	GLUCOSE	UREA	CREATININE	SODIUM	POTTASSIUM	TOTAL COUNT	NEUTROPHIL %	LYMPHOCYT %	HEMOGLOBIN %	PERITONITIS	ASA SCORE	DURATION between PAIN & SURGERY in HOURS	DURATION between ADMISSION & SURGERY in HOURS	SURGERY	perforation SITE	SIZE in CM	CONTAMINATION in LITRES	CONTENTS	PROCEDURE	DURATION OF SURGERY in hours
51	Ramadoss	107	68	1.7	141	4.2	15700	74	15	11	Y	2	33	3.3	O	PP	0.5	0.5	B	MG	2
52	Lakshmanan	96	22	0.8	137	4.6	9200	90	9	13	Y	2	32	7.3	O	A	0.5	1	B	MG	1.3
53	Vishalkumar	96	69	2	145	5.1	2500	56	15	8	Y	3	52	2.45	O	A	0.5	1	B	MG	2.3
54	Krishnapillai	86	65	2.1	146	4.8	7800	90	9	9	Y	3	57	2.3	O	D1	3	2	GAN	MG	1.3
55	Raj	126	125	1.5	137	3.6	6900	91	6	12	Y	3	28	4	O	P	0.5	0.5	PUS	G	2.3
56	Radhakrishnan	110	28	1.1	140	4	9800	78	25	9	Y	2	10 DAYS	12	O	D1	0.5	0	B	G	2.3
57	Appuraj	126	36	1	142	3.9	10400	72	14	11	Y	2	58	8	O	D1	0.5	0.05	B	MG	2
58	Purusothaman	110	26	0.9	131	3.6	7700	70	20	12	Y	2	75	2	O	D1	0.5	1.5	B	MG	2
59	Gopal	68	132	4.2	138	5.4	14400	74	24	11	Y	3	82	10	O	A	1.5	0.8	PUS	G	2.5
60	Raji	106	106	3.2	130	5.9	15600	90	9	8	Y	5	96	13	O	A	1	2	B	G	2
61	Ravikumar	90	56	2.1	140	3.7	12000	88	13	8	Y	3	7 days	3	O	A	0.5	3	B	MG	3
62	Gnanamoorthy	70	101	5.1	139	3	6400	90	7	11	Y	3	72	11	O	A	1	3	B	G	2
63	Moorthy	91	74	1.6	136	3.5	7200	60	35	13	Y	2	7 days	7	O	PP	0.5	1	B	MG	1.3
64	Yesuraj	98	28	0.8	138	3.7	11600	60	38	9.8	Y	2	26	1.3	O	D1	0.5	0.5	B	MG	2
65	Jeyaraman	114	44	1.1	139	3.3	9500	83	12	13	Y	2	52	5	O	D1	0.5	2.5	B	G	2
66	Balaji	60	34	1.1	140	3.4	7500	84	9	11	Y	2	53	5	O	D1	1	2	B	G	2
67	Lingaiyan	87	40	1	131	3.8	3700	90	8	13	Y	2	76	4.3	O	D1	0.3	0.8	B	G	2.3
68	Srinivasan	153	42	0.7	132	4.8	3400	60	34	13	Y	2	30	7	O	D1	0.5	2	B	G	1.5
69	Bindy	114	40	0.9	128	4.6	5600	90	7	12	Y	2	17	5	O	D1	1	0.5	B	G	1.3
70	Shankar	90	45	2.1	136	4.6	11600	78	20	11	Y	2	39	3	O	A	0.5	1	B	G	1.3
71	Sabiq	69	98	3.2	142	5.8	6800	90	8	9.6	Y	2	30	18	O	PP	0.5	0.75	B	MG	1.3
72	Rangan	90	38	1	137	3.8	10400	68	30	11	Y	2	45	9	O	D1	0.5	1	B	G	1.3
73	Kesavan	108	28	0.9	138	3.8	7100	77	15	10	Y	2	41	5	O	D1	1	1	B	G	2
74	Premkumar	108	26	0.9	135	3.6	10200	82	12	12	Y	2	30	5	O	D1	0.5	1	B	G	1
75	Ahamed	110	28	1.2	134	3	11000	63	30	12	Y	2	42	6	O	P	1	1	B	MG	2

S.No	NAME	GLUCOSE	UREA	CREATININE	SODIUM	POTTASSIUM	TOTAL COUNT	NEUTROPHIL %	LYMPHOCYT %	HEMOGLOBIN %	PERITONITIS	ASA SCORE	DURATION between PAIN & SURGERY in HOURS	DURATION between ADMISSION & SURGERY in HOURS	SURGERY	perforation SITE	SIZE in CM	CONTAMINATION in LITRES	CONTENTS	PROCEDURE	DURATION OF SURGERY in hours
76	Manoharan	106	46	1.1	137	3.6	10600	90	9	11	Y	2	27	3	O	D1	0.5	1	B	G	1.3
77	Chandran	120	28	1.5	133	4.6	8100	80	12	12	N	2	51	3.15	O	D1	0.3	0.1	B	G	1.3
78	Tarunkumar	94	25	0.8	138	3.8	9700	76	20	12	N	1	31	7.45	O	A	0.4	0.3	B	MG	1.45
79	Rajendran	132	28	0.8	138	4.8	11400	93	7	16	Y	1	76	3	O	D1	0.4	0.5	B	G	1.45
80	Suriyakumar	82	64	2.5	136	4	4000	96	5	14	Y	1	53	5	O	A	0.5	3	B	G	1.3
81	Sivaraj	98	28	0.9	133	3.7	10100	86	10	14	Y	1	52	4	O	A	0.5	0.5	B	MG	1.45
82	Gokul	80	44	1.3	142	3.8	10000	72	23	12	N	1	56	8	O	D1	0.5	0.3	B	MG	1.3
83	Pandian	132	30	1,3	139	3.2	5000	57	37	14	Y	1	75	4	O	D1	0.5	0.5	B	G	1.3
84	Manimaran	185	23	0.7	136	2.5	5900	63	33	13	Y	1	52	2	O	D1	1	1.5	B	MG	2
85	Sandhanagopal	106	28	0.7	136	3.6	10500	72	27	11	Y	1	28	3.3	O	A	1	2	B	G	2
86	Ganesan	96	36	1.5	142	4.1	5800	84	14	9.2	Y	1	12	5.3	O	D1	0.5	0.2	B	G	2
87	Ganapathy	80	69	4.3	143	4	5500	60	35	6.3	Y	3	72	5.3	O	D1	0.5	2	B	MG	1.3
88	Muruganantham	90	67	3	145	4	7800	89	11	7	Y	4	24	6	O	-	-	2	B	drain	1
89	Barani	83	28	0.8	140	3.8	7200	88	10	11	Y	2	28	4	O	P	0.5	0.2	B	MG	1.2
90	Nithyanandham	114	28	0.9	134	3.5	2600	70	29	16	Y	2	26	2	O	D1	0.7	1.5	B	MG	2
91	Rajesh	120	28	0.8	141	3	12100	80	10	9.6	Y	3	80	8	O	PP	0.3	1	B	MG	1.3
92	Ramkumar	59	34	1.2	143	3.6	19700	92	6	13	Y	1	29	5.3	O	P	0.4	1	B	MG	2
93	Abdulla	67	155	2.9	136	5.2	15900	92	4	9.6	Y	2	30	5	O	D1	2	3	B	G	2
94	Anduraj	107	68	1.7	141	4.2	15700	74	15	11	Y	2	33	3.3	O	PP	0.5	0.5	B	MG	2
95	Prabakaran	96	22	0.8	137	4.6	9200	90	9	13	Y	2	32	7.3	O	A	0.5	1	B	MG	1.3
96	Rajan	96	69	2	145	5.1	2500	56	15	8	Y	3	52	2.45	O	A	0.5	1	B	MG	2.3
97	Smith	86	65	2.1	146	4.8	7800	90	9	9	Y	3	57	2.3	O	D1	3	2	B	MG	1.3
98	Devanathan	126	125	1.5	137	3.6	6900	91	6	12	Y	3	28	4	O	P	0.5	0.5	PUS	G	2.3
99	Mani	110	28	1.1	140	4	9800	78	25	9	Y	2	10 DAYS	12	O	D1	0.5	0	B	G	2.3
100	Periyasamy	126	36	1	142	3.9	10400	72	14	11	Y	2	58	8	O	D1	0.5	0.05	B	MG	2

S.No	NAME	RESPIRATORY SUPPORT	CIRCULATORY SUPPORT	RENAL FUNCTION	perforation LEAK	FLUID COLLECTION	PARALYTIC ILEUS	INTESTINAL OBSTRUCTION	BLEEDING	WOUND INFECTION	Wound dehiscence	RESPIRATORY complication	CARDIAC Complication	RENAL Complication	HEPATIC complication	MODS	FINAL OUTCOME	HOSPITAL STAY in DAYS	CAUSE OF DEATH
1	Dhamodaran	N	N	A	N	N	N	N	N	N	N	LRI	N	N	N	N	Discharge	11	-
2	Arasu	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	10	-
3	Venkatesh	Y	Y	NA								ARDS	ARREST	AKI	N	Y	Death	1	Septic shock
4	Joseph	Y	Y	NA								ARDS	ARREST	AKI	N	Y	Death	1	Septic shock
5	Subramani	N	N	A	N	N	N	N	N	N	N	N	N	AKI	N	N	Discharge	10	
6	Hussain	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	13	
7	Narayanasamy	N	N	A	N	N	Y	N	N	N	N	LRI	N	N	N	N	Discharge	14	
8	Maruthu	n	n	A	n	n	n	n	n	n	n	n	n	n	n	n	Discharge	9	
9	Paneer selvam	Y	Y	NA	N	N	n	n	n	Y	n	LRI	n	AKI	n	n	Death	6	AKI
10	Kanimozhi	y	y	NA	n	n	n	n	n	y	n	n	n	n	N	Y	Death	5	MODS
11	Palani	y	y	NA	n	n	n	n	n	Y	y	n	N	prerenal	n	n	Death	1	Septic shock
12	Krishnamoorthy	y	y	A	n	n	n	n	n	y	y	PE	N	prerenal	n	n	Discharge	16	
13	Selvam	y	N	A	n	n	n	n	n	Y	Y	PE,LRI	N	prerenal	n	n	Discharge	18	
14	Murugesan	n	n	A	n	n	n	n	n	n	n	n	N	n	n	n	Discharge	10	
15	Muna	n	n	A	n	Y	n	n	n	Y	y	PE,LRI	N	n	n	n	Discharge	23	
16	Sriram	n	n	A	n	n	n	n	n	n	n	N	N	n	n	n	Discharge	13	
17	Nagendran	n	n	A	n	n	n	n	n	Y	Y	N	N	n	n	n	Discharge	15	
18	Kamaraj	y	n	A	n	n	n	n	n	y	y	LRI	N	n	n	n	Discharge	11	
19	Sighumalick	n	n	A	n	n	n	n	n	y	n	N	N	n	n	n	Discharge	10	
20	Mani	Y	n	A	n	n	n	n	n	n	N	ARDS	N	n	n	n	Death	3	Septic shock
21	Rangachari	Y	n	NA	n	n	n	n	n	y	N	LRI	N	Renal	n	n	Death	10	AKI
22	Pintu	n	n	A	n	n	n	n	n	n	n	N	N	n	n	n	Discharge	8	
23	Kumar	n	n	A	n	n	n	n	n	n	n	N	N	n	n	n	Discharge	8	
24	Siva	n	n	A	n	n	n	n	n	n	n	N	N	n	n	n	Discharge	13	
25	Arulraj	n	n	A	n	Y	Y	n	n	n	N	PE	N	n	n	n	Discharge	18	

S.No	NAME	RESPIRATORY SUPPORT	CIRCULATORY SUPPORT	RENAL FUNCTION	perforation LEAK	FLUID COLLECTION	PARALYTIC ILEUS	OBSTRUCTION	BLEEDING	INFECTION	Wound dehiscence	RESPIRATORY complication	CARDIAC Complication	RENAL Complication	HEPATIC complication	MODS	FINAL OUTCOME	HOSPITAL STAY in DAYS	CAUSE OF DEATH
26	Kumar	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	9	
27	Paramasivam	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	9	
28	Masilamani	N	N	A	N	N	N	N	N	Y	N	N	N	N	N	N	Discharge	11	
29	Prakash	N	N	A	N	N	N	N	N	Y	Y	N	N	N	N	N	Discharge	25	
30	Saravanan	Y	Y	NA	N	N	N	N	N	Y	N	Y	Y	AKI	N	N	Discharge	30	
31	Sivakumar	N	N	A	N	N	N	N	N	Y	N	N	N	N	N	N	Discharge	14	
32	Abshiek kumar	N	N	A	N	N	N	N	N	Y	N	N	N	N	N	N	Discharge	9	
33	Gangan	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	11	
34	Elumalai	N	N	A	N	N	N	N	N	Y	N	N	N	N	N	N	Discharge	13	
35	Manikandan	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	10	
36	Kumar	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	10	
37	Ettiyappan	Y	Y	NA	N	N	N	N	N	N	N	LRI	N	RENAL	N	Y	Death	7	MODS
38	Devaraj	Y	Y	NA	-	-	-	-	-	-	-	-	-	RENAL	-	Y	Death	1	Septic shock
39	Babu	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	10	
40	Murugan	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	14	
41	Santhanam	N	N	A	N	N	N	N	N	Y	N	N	N	N	N	N	Discharge	10	
42	Arumugam	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	8	
43	Fakrudeen	Y	Y	NA	N	N	N	N	N	N	N	LRI	N	RENAL	N	N	Death	2	Septic shock
44	Anbarasan	N	N	A	N	N	N	N	N	N	N	LRI	N	N	N	N	Discharge	11	
45	Jeyachandran	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	10	
46	Rajagopal	Y	Y	NA								ARDS	ARREST	AKI	N	Y	Death	1	MODS
47	Siva	Y	Y	NA								ARDS	ARREST	AKI	N	Y	Death	1	MODS
48	Kasi	N	N	A	N	N	N	N	N	N	N	N	N	AKI	N	N	Discharge	10	
49	Baskar	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	13	
50	Bala	N	N	A	N	N	Y	N	N	N	N	LRI	N	N	N	N	Discharge	14	

S.No	NAME	RESPIRATORY SUPPORT	CIRCULATORY SUPPORT	RENAL FUNCTION	perforation LEAK	FLUID COLLECTION	PARALYTIC ILEUS	INTESTINAL OBSTRUCTION	BLEEDING	WOUND INFECTION	Wound dehiscence	RESPIRATORY complication	CARDIAC Complication	RENAL Complication	HEPATIC complication	MODS	FINAL OUTCOME	HOSPITAL STAY in DAYS	CAUSE OF DEATH
51	Ramadoss	N	N	A	N	N	N	N	N	N	N	LRI	N	N	N	N	Discharge	11	-
52	Lakshmanan	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	10	-
53	Vishalkumar	Y	Y	NA								ARDS	ARREST	AKI	N	Y	Death	1	MODS
54	Krishnapillai	Y	Y	NA								ARDS	ARREST	AKI	N	Y	Death	1	MODS
55	Raj	N	N	A	N	N	N	N	N	N	N	N	N	AKI	N	N	Discharge	10	
56	Radhakrishnan	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	13	
57	Appuraj	N	N	A	N	N	Y	N	N	N	N	LRI	N	N	N	N	Discharge	14	
58	Purusothaman	n	n	A	n	n	n	n	n	n	n	n	n	n	n	n	Discharge	9	
59	Gopal	Y	Y	NA	N	N	n	n	n	Y	n	LRI	n	AKI	n	n	Death	6	AKI
60	Raji	y	y	NA	n	n	n	n	n	y	n	n	n	n	N	Y	Death	5	MODS
61	Ravikumar	y	y	NA	n	n	n	n	n	Y	y	n	N	prerenal	n	n	Death	1	Septic shock
62	Gnanamoorthy	y	y	A	n	n	n	n	n	y	y	PE	N	prerenal	n	n	Discharge	16	
63	Moorthy	y	N	A	n	n	n	n	n	Y	Y	PE,LRI	N	prerenal	n	n	Discharge	18	
64	Yesuraj	n	n	A	n	n	n	n	n	n	n	n	N	n	n	n	Discharge	10	
65	Jeyaraman	n	n	A	n	Y	n	n	n	Y	y	PE,LRI	N	n	n	n	Discharge	23	
66	Balaji	n	n	A	n	n	n	n	n	n	n	n	N	n	n	n	Discharge	13	
67	Lingaiyan	n	n	A	n	n	n	n	n	Y	Y	n	N	n	n	n	Discharge	15	
68	Srinivasan	y	n	A	n	n	n	n	n	y	y	LRI	N	n	n	n	Discharge	11	
69	Bindy	n	n	A	n	n	n	n	n	y	n	n	N	n	n	n	Discharge	10	
70	Shankar	Y	n	A	n	n	n	n	n	n	N	ARDS	N	n	n	n	Death	3	ARDS
71	Sabiq	Y	n	NA	n	n	n	n	n	y	N	LRI	N	Renal	n	n	Death	10	AKI
72	Rangan	n	n	A	n	n	n	n	n	n	n	n	N	n	n	n	Discharge	8	
73	Kesavan	n	n	A	n	n	n	n	n	n	n	n	N	n	n	n	Discharge	8	
74	Premkumar	n	n	A	n	n	n	n	n	n	n	n	N	n	n	n	Discharge	13	
75	Ahamed	n	n	A	n	Y	Y	n	n	n	N	PE	N	n	n	n	Discharge	18	

S.No	NAME	RESPIRATORY SUPPORT	CIRCULATORY SUPPORT	RENAL FUNCTION	perforation LEAK	FLUID COLLECTION	PARALYTIC ILEUS	INTESTINAL OBSTRUCTION	BLEEDING	WOUND INFECTION	Wound dehiscence	RESPIRATORY complication	CARDIAC Complication	RENAL Complication	HEPATIC complication	MODS	FINAL OUTCOME	HOSPITAL STAY in DAYS	CAUSE OF DEATH
76	Manoharan	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	9	
77	Chandran	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	9	
78	Tarunkumar	N	N	A	N	N	N	N	N	Y	N	N	N	N	N	N	Discharge	11	
79	Rajendran	N	N	A	N	N	N	N	N	Y	Y	N	N	N	N	N	Discharge	25	
80	Suriyakumar	Y	Y	NA	N	N	N	N	N	Y	N	Y	Y	AKI	N	N	Discharge	30	
81	Sivaraj	N	N	A	N	N	N	N	N	Y	N	N	N	N	N	N	Discharge	14	
82	Gokul	N	N	A	N	N	N	N	N	Y	N	N	N	N	N	N	Discharge	9	
83	Pandian	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	11	
84	Manimaran	N	N	A	N	N	N	N	N	Y	N	N	N	N	N	N	Discharge	13	
85	Sandhanagopal	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	10	
86	Ganesan	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	10	
87	Ganapathy	Y	Y	NA	N	N	N	N	N	N	N	LRI	N	RENAL	N	Y	Death	7	MODS
88	Muruganantham	Y	Y	NA	-	-	-	-	-	-	-	-	-	RENAL	-	Y	Death	1	Septic shock
89	Barani	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	10	
90	Nithyanandham	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	14	
91	Rajesh	N	N	A	N	N	N	N	N	Y	N	N	N	N	N	N	Discharge	10	
92	Ramkumar	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	8	
93	Abdulla	Y	Y	NA	N	N	N	N	N	N	N	LRI	N	RENAL	N	N	Death	2	Septic shock
94	Anduraj	N	N	A	N	N	N	N	N	N	N	LRI	N	N	N	N	Discharge	11	
95	Prabakaran	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	10	
96	Rajan	Y	Y	NA								ARDS	ARREST	AKI	N	Y	Death	1	MODS
97	Smith	Y	Y	NA								ARDS	ARREST	AKI	N	Y	Death	1	MODS
98	Devanathan	N	N	A	N	N	N	N	N	N	N	N	N	AKI	N	N	Discharge	10	
99	Mani	N	N	A	N	N	N	N	N	N	N	N	N	N	N	N	Discharge	13	
100	Periyasamy	N	N	A	N	N	Y	N	N	N	N	LRI	N	N	N	N	Discharge	14	